CLINICAL PRESENTATION, MANAGEMENT STRATEGIES, AND IN-HOSPITAL OUTCOME IN CARDIAC RUPTURE

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I hereby declare that the thesis entitled "**CLINICAL PRESENTATION, MANAGEMENT STRATEGIES, AND IN-HOSPITAL OUTCOME IN CARDIAC RUPTURE**" submitted towards partial fulfillment of the requirements for the award of DM in Cardiology degree to BFUHS is a compilation of original work carried out by me under the supervision of Dr. Shibba Takkar Chhabra, Professor, Department of Cardiology, Dr. Naved Aslam, Professor, Department of Cardiology and Dr. Gurbhej Singh, Assistant professor, Department of Cardiology. No part of this thesis has formed the basis for the award of any degree previously.

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Dedicated

to

my Family

&

Teachers

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“As we express our gratitude, we must never forget that the highest appreciation is not to utter words, but to live by them.”

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Dr. Amit Kumar Gupta

LIST OF ABBREVIATIONS

AMI	:	Acute Myocardial Infarction
APEX-AMI	:	Assessment of Pexelizumab in Acute
CABG	:	Coronary Artery By-pass Grafting
CAD	:	Coronary Artery Disease
CAG	:	Coronary angiography
CKD	:	Chronic Kidney Disease
COPD	:	Chronic Obstructive Pulmonary Disease
CVA	:	Cerebrovascular Accident
DVD	:	Double Vessel Disease
ECMO	:	Extracorporeal Membrane Oxygenation
EuroSCORE II	:	European system for cardiac operative risk evaluation II
		for Occluded Coronary Arteries
GRACE	:	Global Registry of Acute Coronary Events
GUSTO-I	:	Global Utilization of Streptokinase and TPA
IABP	:	Intra- Aortic Balloon Pump
LAD	:	Left Anterior Descending Artery
LCX	:	Left Circumflex Artery
LVFWR	:	Left Ventricular Free Wall Rupture
MFWR	:	Myocardial Free Wall Rupture
MIDAS	:	Myocardial Infarction Data Acquisition
		Myocardial Infarction
PAD	:	Peripheral Artery Disease
PPCI	:	Primary Percutaneous Coronary Intervention
RCA	:	Right Coronary Artery
RRT	:	Renal Replacement Therapy
STEMI	:	ST- Elevation Myocardial Infarction
SVD	:	Single Vessel Disease
		System
TIA	:	Transient Ischemic Attack
TVD	:	Triple Vessel Disease
VSR	:	Ventricular Septal Rupture

ABSTRACT

Introduction: Cardiac rupture is a disastrous complication of acute ST-elevation myocardial infarction (STEMI). With the advancement in medical treatment and PPCI, the morbidity and mortality of cardiac rupture after STEMI have decreased. Still Cardiac rupture accounts for approximately 20% of STEMI patient in-hospital deaths and remain a rare and lethal complication of AMI.

Aims & Objectives:

- To study the clinical presentation in patients of cardiac rupture.
- To evaluate the management strategies and in-hospital outcome in patients of cardiac rupture.

Material & Methods: It was an observational study done in retrospective and prospective manner. For retrospective analysis, data of all consecutive patients with cardiac rupture admitted in DMC&H during last 10 years was taken from institutional data base. For prospective analysis, informed consent was taken from patient or patient's attendant presenting to emergency department of DMC&H with cardiac rupture from 1st August 2021 to 31 December 2022. Patients were assessed on basis of history, physical examination, investigation and treatment received. The data was collected in case record form & evaluated.

Results: Out of 82 patients of cardiac rupture, 48 had ventricular septum rupture while 34 had free wall rupture. On statistical analysis, age > 70 years (p value =0.004), early rupture (</=2days) (p value =0.004), Killip class IV on admission (p value =0.035), elevated TLC (p value=0.015), higher troponin-T (p value =0.001), conservative management (p value =0.001) were associated with in-hospital mortality, while use of IABP (p value =0.032) & Surgical repair (p value =0.001) were associated with in-hospital survival in patients of cardiac rupture.

Conclusion: Patients with cardiac rupture are more likely to be elderly > 65 yrs, hypertensive, have no prior history of CAD, first MI, usually anterior wall MI, have SVD, have lower rate of PPCI, and have higher in-hospital mortality. Compared with survivors, non-survivors are older >70 yrs, have early occurrence of cardiac rupture(</=2days), are in killip class IV on admission, have elevated TLC & higher troponin-T, and lower rate of surgical repair. Role of IABP in survival remains debatable. Surgical repair remains the most valuable factor associated with survival in patients of cardiac rupture.

Keywords: STEMI, LVFWR, VSR, PPCI, CABG, IABP

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INTRODUCTION

Cardiac rupture is a disastrous complication of acute ST-elevation myocardial infarction (STEMI). It is the second-most-common cause of in-hospital death in STEMI patients. The incidence of cardiac rupture and mortality in STEMI are high post-thrombolysis which accelerates cardiac rupture in the acute phase [1,2]. The incidence of cardiac rupture after STEMI was 6% during thrombolytic therapy era or the no reperfusion era [1].

In recent years with the widespread use of primary percutaneous coronary intervention (PPCI) the incidence of cardiac rupture after acute myocardial infarction has decreased to less than 2%. With the advancement in medical treatment and PPCI, the morbidity and mortality of cardiac rupture after STEMI have decreased [3]. Still Cardiac rupture accounts for approximately 20% of STEMI patient in-hospital deaths and remains a rare and lethal complication of AMI [4].

The diagnosis of cardiac rupture is generally based on physical examination, changes in vital signs, clinical suspicion and further can be confirmed with 2D Echocardiography.[5] Generally, cardiac rupture are classified into three types based on the site of the rupture: rupture of the ventricular free wall, of the interventricular septum and of the papillary muscles.[6] Normally cardiac rupture occurs in about half of cases within the first 5 days and in over 90% of patients within 2 weeks after onset of acute myocardial infarction.[7–9]

Traditional risk factors for cardiac rupture includes advanced age, female, absence of prior angina, history of hypertension, no history of myocardial infarction, anterior infarct, absence of PPCI, single-vessel disease without collateral circulation, anemia, and elevated heart rate [3,10,11].

Clinical presentation and course are variable, depending upon the location, the size and the time-course of the rupture [12]. It varies from a catastrophic blowout type characterized by cardiogenic shock and eventually cardiac arrest, to the oozing type with hemodynamic instability and pericardial effusion [13].

Clinically cardiac ruptures can be divided into: acute rupture which results in death within a few minutes (<30 min) due to massive hemorrhage into the pericardial cavity; subacute rupture which is characterized by a

smaller tear that might be temporarily sealed by a clot or fibrinous pericardial adhesions which may be compatible with life for several hours or even longer; chronic rupture with false aneurysm formation which occurs when the leakage of blood is slow and when surrounding pressure on the epicardium temporarily controls the hemorrhage. Adhesions form between the epicardium and pericardium which reinforce and contain the rupture [14].

Prodromal manifestations include persistent chest pain, intractable vomiting, restlessness, also electrocardiogram (ECG) signs as persistent S-T segment elevation, and positive T wave deflection that persists for 72 hours after the onset of chest pain [8,15].

In most cases classical signs of cardiac tamponade, including pulsus paradoxus and diastolic pressure equalization don't exist [9].

Cardiac rupture must be suspected in patients of recent myocardial infarction who have recurrent or persistent chest pain, sometimes of the pericardial type, hemodynamic instability, syncope (resulting from transient electromechanical dissociation), and pericardial tamponade, New ST segment changes ("saddle shaped ST-segment elevation") [7,16].

Emergency bedside transthoracic and/or contrasted trans-esophageal echocardiograms are considered the gold standard diagnostic tools [17], for timely diagnosis of cardiac rupture and have a sensitivity and specificity of more than 90% [18].

Timely diagnosis and emergent treatment is essential for saving patient's lives. The median time of death following LVFWR is 8 hours [18]. Therefore, a high index of suspicion and close monitoring of patient's symptoms and signs is necessary.

In the immediate setting the treatment for cardiac rupture rupture is generally supportive (conservative strategy) and surgical correction of the rupture is recommended as early as feasible. Conservative management includes maintaining hemodynamic stability, prolonged bed rest, strict blood pressure control, and insertion of an IABP [19]. Figure 1 & 2 demonstrate approach for management of different types of cardiac rupture.

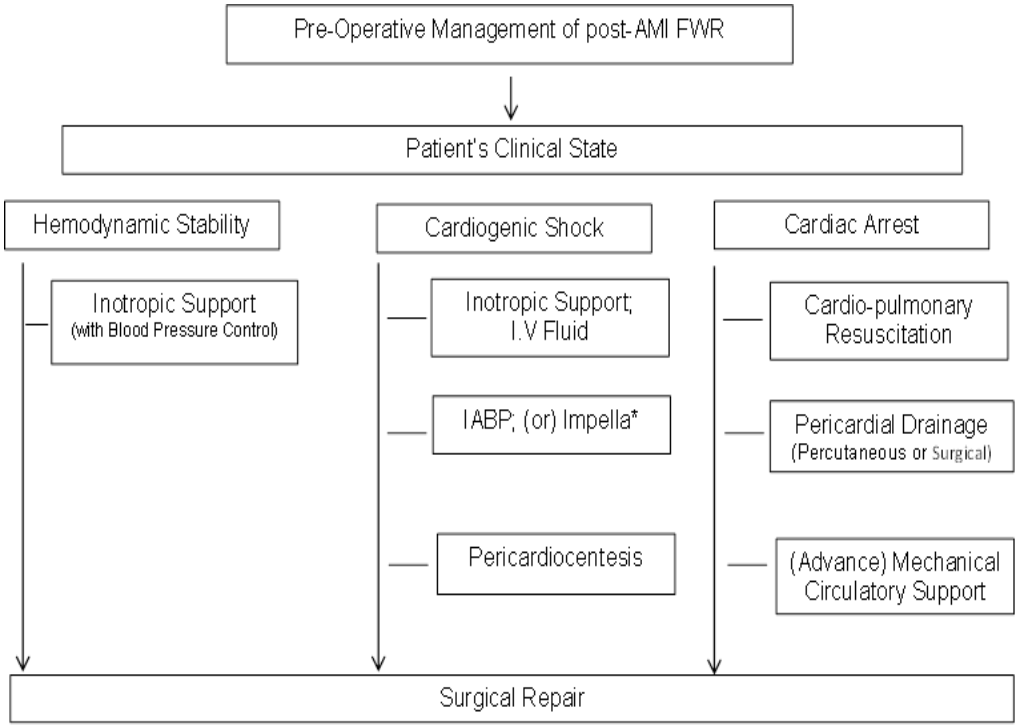

Figure 1. A Multidisciplinary approach for managing left ventricular free-wall rupture.

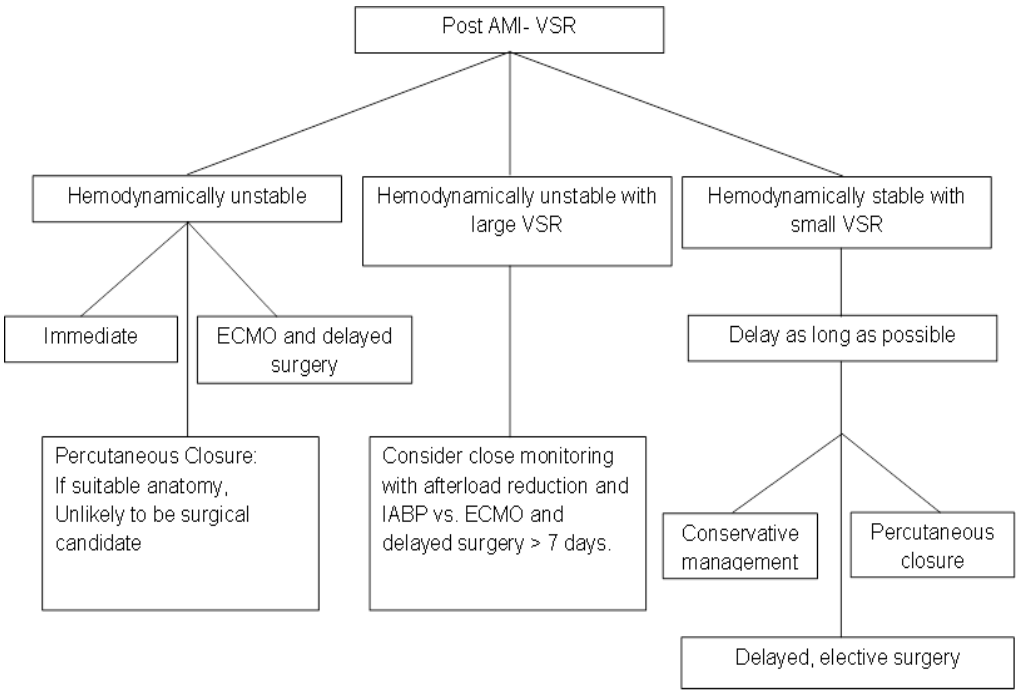

Figure 2: A Multidisciplinary approach for managing acute ventricular septal rupture.

The survival rate among patients of AMI with conservatively treated cardiac rupture was reported to be only 10%. Due to the poor prognosis of conservative strategies [20], surgical repair is typically the cornerstone of the treatment strategy. Despite different surgical approaches the in-hospital mortality in patients of cardiac rupture continues to remain high [21,22]. The mortality rate for free-wall rupture is 75% to 90%, and the mortality rate for ventricular septal rupture is 40% to 75% [10].

As a rare but fatal complication of STEMI, the prognosis of cardiac rupture remains poor, and it could lead to sudden and devastating death in patients after myocardial infarction (MI). However, due to its rare and unpredictable incidence, there is lack of evidence for its clinical characteristics. Moreover, as the sample sizes are generally limited in the previous studies, the effect of cardiac rupture on mortality has not been studied well and has varied among different studies, possibly due to several factors including diverse reperfusion strategies for STEMI, various cardiac rupture sites, time to diagnosis and different treatment strategies. Thus, this study aimed to evaluate the clinical presentation, management strategies, and in-hospital outcomes in patients of cardiac rupture in a tertiary care centre in north India.

AIMS AND OBJECTIVES

- To study the clinical presentation in patients of cardiac rupture.
- To evaluate the management strategies and in-hospital outcome in patients of cardiac rupture.

REVIEW OF LITERATURE

History: The first free-wall rupture of the heart after AMI was described by William Harvey in 1647.[23] In 1769, Morgagni collected and reported 11 cases of myocardial rupture discovered postmortem.[24] Ironically, Morgagni himself later died of ventricular rupture. However, it was not until 1925, in the report of Krumbhaar and Crowell, that the relationship of LVFWR to myocardial infarction was first pointed out.[25]

Hatcher and associates, from Emory University, in 1970 described the first successful operation for free-wall rupture of the right ventricle.[26] Two years later, Fitzgibbon et al. reported the successful repair of LVFWR associated with ischemic heart disease.[27]

In 1975 Becker and Mantgem classified cardiac rupture into three types. Type I rupture shows an abrupt, slit-like tear and is associated with acute infarcts, 24 hr in age. Type II rupture demonstrates erosion of the infarcted myocardium, and correlates clinically with a sub-acute presentation. Type III rupture exhibits concomitant aneurysm formation with significant thinning of the septum and subsequent rupture, a process associated with older infarcts [28].

Honan MB et al (1990) examined the relation between the risk of cardiac rupture and the timing of thrombolytic therapy for acute myocardial infarction. To test the hypothesis that cardiac rupture is prevented by early thrombolytic therapy but is promoted by late treatment, randomized controlled trials of thrombolytic agents for myocardial infarction were pooled. A logistic regression model including 58 cases of cardiac rupture among 1,638 patients from four trials showed that the odds ratio (treated/control) of cardiac rupture was directly correlated with time to treatment ($p = 0.01$); at 7 h, the odds ratio was 0.4 (95% confidence limits 0.17 to 0.93); at 11 h, it was 0.93 (0.53 to 1.60) and at 17 h, it was 3.21 (1.10 to 10.1). They concluded that thrombolytic therapy early after acute myocardial infarction improves survival and decreases the risk of cardiac rupture. Late administration of thrombolytic therapy also appears to improve survival but may increase the risk of cardiac rupture [29].

In a study from united states Becker RC et al (1996) studied the incidence, timing and prevalence as a cause of death from cardiac rupture in patients of acute myocardial infarction. They enrolled 350,755

patients,122,243 received thrombolytic therapy. In-hospital mortality for the overall patient population, those not treated with thrombolytics (n = 228,512) and those given thrombolytics were 10.4%, 12.9% and 5.9%, respectively (p<0.001). Cardiogenic shock was the most common cause of death in each patient group. Although the incidence of cardiac rupture was low (<1.0%), it was responsible for 7.3%, 6.1% and 12.1%, respectively, of in-hospital deaths (p<0.001). Death from rupture occurred earlier in patients given thrombolytic therapy, with a clustering of events within 24 h of drug administration. Despite the early risk, death rates were comparatively low in thrombolytic-treated patients on each of the first 30 days. By multivariable analysis, thrombolytics, prior myocardial infarction, advancing age, female gender and intravenous beta-blocker use were independently associated with cardiac rupture [2].

Crenshaw BS et al (2000) compared enrollment characteristics, angiographic patterns, and outcomes (30-day and 1-year mortality) of patients enrolled in the Global Utilization of Streptokinase and TPA for Occluded Coronary Arteries (GUSTO-I) trial with and without a confirmed diagnosis of VSD. In all 84 of the 41021 patients (0.2%) developed VSD, a smaller percentage than reported in the pre-thrombolytic era. The median time from symptom onset to VSD diagnosis was 1 day. Enrollment factors most associated with this complication were advanced age, anterior infarction, female sex, and no previous smoking. The infarct artery was more often the left anterior descending and more likely to be totally occluded in patients who developed VSD. Mortality at 30 days was higher in patients with VSD than in those without this complication (73.8% versus 6.8%, P<0.001). Patients with VSD selected for surgical repair (n=34) had better outcomes than patients treated medically (n=35; 30-day mortality, 47% versus 94%), and the patients who undergo thrombolysis within 6 hours of infarction onset may have a reduced risk of later VSD. If patients develop this mechanical complication, however, it typically occurs sooner than described in the pre-thrombolytic era. Despite improvements in medical therapy and percutaneous and surgical techniques, mortality with this complication remains extremely high [30].

In a study from Israel Solodky A et al (2001) compared the incidence of cardiac rupture among patients with acute myocardial infarction treated by thrombolysis versus percutaneous transluminal coronary angioplasty. During the 5-month period, 2,377 consecutive patients were hospitalized with the diagnosis of AMI. Of these, 103 (4%) underwent primary PTCA, 1,162 (49%)

patients received thrombolytic therapy, and 1,112 (47%) did not receive acute reperfusion therapy. They found that advanced age, female gender, hypotension, tachycardia, presence of heart failure on admission (Killip class > I), and thrombolytic therapy were independently associated with the risk of cardiac rupture after AMI.[31]

Ikeda N et al [2004] studied the effect of reperfusion therapy on cardiac rupture after myocardial infarction in Japanese. Among 1,296 consecutive AMI patients Cardiac rupture occurred in 45 patients (3.5%), comprising left ventricular free wall rupture (n=23), ventricular septal perforation (n=20) and papillary muscle rupture (n=2). Early-phase rupture (within 72 h after AMI onset) was associated with anterior infarction. Of the 45 patients who experienced ruptures, 24 (53.3%) survived to discharge; 21 (46.6%) of the 45 ruptures occurred after admission. Successful reperfusion therapy was performed within 24 h for 840 patients. The incidence of rupture was significantly lower in reperfused patients than non-reperfused patients (0.6% vs 3.5%, respectively; p<0.01). Peak C-reactive protein concentration was a reliable predictor of late-phase rupture (p=0.01), but not of early-phase rupture [32].

Figueras J et al (2008) investigated Changes in hospital mortality rates in 425 patients with acute ST-elevation myocardial infarction and cardiac rupture over a 30-year period divided into 5 intervals (1977 to 1982, 1983 to 1988, 1989 to 1994, 1995 to 2000, and 2001 to 2006). Of a total of 6678 consecutive patients, 425 experienced a free wall rupture (280 with cardiac tamponade: 227 with electromechanical dissociation and 53 with hypotension) or a septal rupture (145). After the exclusion of referrals from other centers (n=44), the incidence of definite cardiac rupture (septal rupture, anatomic evidence of free wall rupture, or electromechanical dissociation) declined progressively (6.2% in 1977 to 1982 to 3.2% in 2001 to 2006; P<0.001) in parallel with a progressive use of reperfusion therapy (0% to 75.1%; P<0.001). In addition, among patients with cardiac rupture, there was a progressive fall in the rate of death (94% to 75%; P<0.001) despite a trend toward increasing age (66+/-8 to 75+/-8 years; P<0.054) in conjunction with better control of systolic blood pressure at 24 hours (130+/-24 versus 110+/-18 mm Hg; P<0.001); an increased use of reperfusion therapy (0% to 59%; P<0.001), beta-blockers (0% to 45%; P<0.001), angiotensin-converting

enzyme inhibitors (0% to 38%; P<0.001), and aspirin (0% to 96%; P<0.001); and a lower use of heparin (99% to 67%; P<0.001) [1].

French JK et al (2010) examined the rates and outcomes of individual mechanical complications of STEMI in the Assessment of Pexelizumab in Acute Myocardial Infarction (APEX-AMI) trial. The APEX-AMI trial recruited patients with STEMI from 17 countries and 296 sites from July 2004 to May 2006. Mechanical complications occurred in 52 of 5,745 patients (0.91%) in the largest reported randomized trial in which primary PCI was the reperfusion strategy. The frequencies were 0.52% (30) for cardiac free-wall rupture (tamponade), 0.17% (10) for ventricular septal rupture, and 0.26% (15) for papillary muscle rupture (3 patients had 2 complications). Ninety-day survival rates were 37% (11) for cardiac free-wall rupture, 20% (2) for ventricular septal rupture, and 73.3% (11) for papillary muscle rupture. These mechanical complications occurred at a median of 23.5 hours after symptom onset and were associated with 44% (23 of 52) survival through 90 days, which accounted for 11% of the 90-day mortality. Factors associated with mechanical complications were older age, female gender, Q waves, presence of radiologic pulmonary oedema, and increased pre-randomization troponin levels. In conclusion, it was found that rates of mechanical complications are lower with primary PCI than those previously reported after fibrinolytic therapy[33].

In 2010 Moreyra AE et al from MIDAS Study Group investigated occurrence and mortality rates in patients with first AMI with VSR (n = 408) and without VSR (n = 148,473) who were hospitalized from 1990 to 2007 using the New Jersey Myocardial Infarction Data Acquisition System (MIDAS) database. The annual rate of VSR in AMI was 0.25% to 0.31%. Compared to patients with AMI without VSR, patients with VSR were older, more likely to be women, had increased rate of chronic renal disease, congestive heart failure, and cardiogenic shock, and were less likely to be hypertensive or diabetic (all p values < 0.0001). During the 18-year study period, we found no change in hospital and 1-year mortalities, which were 41% and 60% in 1990 to 1992 and 44% and 56% in 2005 to 2007, respectively. The survival benefit associated with VSR surgical repair was seen only in hospital (hazard ratio 0.66, 95% confidence interval 0.45 to 0.95) but not at 30 days or 1 year. In conclusion, despite improvement in medical treatment and revascularization techniques, the rate of VSR complicating AMI has not changed during the

previous 2 decades, and the mortality associated with VSR has remained high and relatively constant.[34]

In 2010 Global Registry of Acute Coronary Events (GRACE) Investigators studied factors related to heart rupture in acute coronary syndromes in 60,198 patients and reported that 273 patients had heart rupture (free wall rupture in 118 and ventricular septal rupture in 155). Incidence was 0.9% for ST-segment elevation myocardial infarction (STEMI), 0.17% for non-STEMI, and 0.25% for unstable angina. Hospital mortality was 58 vs. 4.5% in patients without heart rupture. The incidence was lower in STEMI patients with primary percutaneous coronary intervention (PCI) than in those without, but primary PCI was not independently related to heart rupture. Independent variables associated with heart rupture included: ST-segment elevation (STE)/left bundle branch block; ST-segment deviation; female sex; previous stroke; positive initial cardiac biomarkers; older age; higher heart rate; systolic blood pressure/30 mmHg decrease. Conversely, previous MI and the use of low-molecular-weight heparin and beta-blockers during first 24 hr were identified as protective factors for heart rupture.[35]

Honda S et al (2014) evaluated the trends in the clinical and pathological characteristics of cardiac rupture in patients with acute myocardial infarction over 35 years. They divided patients into 3 cohorts: 1977-1989, 1990-2000, and 2001-2011. Of a total of 5699 patients, 144 were diagnosed with cardiac rupture and 45 survived. Over the years, the incidence of cardiac rupture has decreased (1977-1989, 3.3%; 1990-2000, 2.8%; 2001-2011, 1.7%; P=0.002) in association with the widespread use of reperfusion therapy. The mortality rate of cardiac rupture decreased (1977-1989, 90%; 1990-2000, 56%; 2001-2011, 50%; P=0.002) in association with an increase in the rate of emergent surgery. In multivariable analysis, first myocardial infarction, anterior infarct, female sex, hypertension, and age >70 years were significant risk factors for cardiac rupture, whereas impact of hypertension on cardiac rupture has decreased from 2001 to 2011. Primary percutaneous coronary intervention (PPCI) was a significant protective factor against cardiac rupture. In 64 autopsy cases with cardiac rupture, myocardial hemorrhage occurred more frequently in those who underwent PPCI or fibrinolysis than those who did not receive reperfusion therapy (no reperfusion therapy, 18.0%; fibrinolysis, 71.4%; PPCI, 83.3%; P=0.001) [3].

In a retrospective study Nozoe M et al (2014) retrospectively analysed Clinical manifestation of early phase left ventricular rupture complicating acute myocardial infarction in the primary PCI era. Among 1290 AMI patients Ventricular septal rupture (VSR) occurred in 19 cases (1.5%) and left ventricular free wall rupture (LVFWR) in 17 cases (1.3%). Mean observation periods from onset to VSR and LVFWR were 2.6 days. They demonstrated that risk factors for LV rupture were advanced age, female sex, absence of history of angina or myocardial infarction, lack of previous PCI, and absence of previous hypertension. Coronary angiography revealed that the culprit lesions of the left anterior descending artery or single vessel disease were the risk factors for LV rupture. Furthermore, 9 patients (47.4%) with VSR and 8 patients (47.1%) with LVFWR developed LV rupture within 24h after symptoms onset (early rupture). The early rupture demonstrated extremely poor outcome compared with late rupture (in-hospital mortality was 88.2% in early rupture and 63.1% in late rupture) [36].

In another retrospective study Huang SM et al (2015) analysed data from 47 consecutive patients (mean age, 68 ± 9.5 years) with post-infarction VSR who underwent surgical repair to identify independent predictors of postoperative mortality. Percutaneous coronary intervention was performed in 17 (36.2%) patients, intra-aortic balloon pump (IABP) was used in 34 (72.3%), and six (12.8%) were supported with extracorporeal membrane oxygenation (ECMO) preoperatively. Forty-one (87.2%) patients received emergent surgical treatment. Concomitant coronary artery bypass grafting was performed in 27 (57.4%) patients. Operative mortality was 36.2% (17 of 47). The survival rate was 59.3% with concomitant CABG and 70% without concomitant CABG ($p = 14$). Multivariate analysis revealed that the survivors had higher preoperative left ventricular ejection fraction (LVEF) compared with those who died (51% vs. 36.6% $p < 0.001$) and lower European system for cardiac operative risk evaluation II (EuroSCORE II) (22.9 vs. 38.3 $p < 0.001$). The patients receiving total revascularization has long term survival benefit ($p = 0.028$). The EuroSCORE II can be used for an approximate prediction of operative mortality. Preserved LVEF was associated with better prognosis, while the need for postoperative renal replacement therapy was associated with higher early and late mortality [37].

Leitman M et al (2016) evaluated 2,380 patients admitted to Assaf Harofeh Medical Center with the diagnosis of acute myocardial infarction

between January 2008 and February 2015 and reported that 12 (0.5%) developed myocardial rupture. The mean age was 78 years, and there were 7 males and 5 females. Ten patients already had pericardial effusion on admission. Seven patients underwent coronary angiography, while primary percutaneous intervention was performed in 4 patients. Six patients entered the operating room and all survived the procedure. All patients who were treated conservatively died due to rupture. Factors related to the treatment strategy were advanced age (≥ 90 years) and cognitive impairment. The risk of myocardial rupture may be diminished by primary coronary intervention during myocardial infarction, but mortality remains high. An early, comprehensive echocardiographic examination and rapid surgery may contribute to improved survival [38].

Formica F et al (2018) conducted a retrospective study of 35 patients who underwent surgery for LVFWR between January 2000 and December 2016. The mean age of patients was 68.3 years. The in-hospital survival was 65.7% (n = 23), and 13% of survived patients presented with cardiac arrest. The following characteristics were associated with in-hospital mortality at univariable analysis: pre-existing hypertension ($p = 0.02$), need for inotropes ($p = 0.02$) and cardiac arrest ($p < 0.0001$) at presentation, cardiopulmonary resuscitation ($p = 0.004$), preoperative extracorporeal membrane oxygenation ($p = 0.004$), technique of LVFWR repair ($p = 0.013$), operation on extracorporeal membrane oxygenation ($p = 0.005$) and postoperative extracorporeal membrane oxygenation ($p = 0.001$). On multivariable analysis, cardiac arrest at presentation was an independent predictor of in-hospital mortality ($p = 0.003$). The overall mean postoperative follow-up was 8.3 ± 1.3 years. Overall survival rates at 5 and 10 years were 53.2 ± 8.6% and 49.1 ± 8.9%, respectively. Among the survivors, only 6 (26.1%) patients died during follow-up with a 5-year and 10-year overall survival rate of 80.9 ± 8.7% and 74.7 ± 10%, respectively [21].

Varghese S et al (2019) retrospectively studied 5143 patients with AMI at the Central Hospital, Bad Berka. Out of these, seven patients with LVFWR were identified with an overall incidence of 0.14%. Clinically, LVFWR patients presented late to admission since symptom onset (median 24 h vs. 6.1 h; $p < 0.0001$), were more likely in cardiogenic shock (28.6% vs. 3.2%; $p = 0.02$) and were usually accompanied by emergency physicians (71.4% vs. 20.7%; $p = 0.006$), had higher troponin T (median 8.6 vs. 0.5 ng/ml; $p < 0.0002$),

higher CRP (median 50 vs. 0.5 mg/l; p = 0.05) as well as a lower haematocrit values (0.33 vs. 0.42; p = 0.04). All LVFWR patients were operated (100% vs. 1.6%; p < 0.001). The patients had lower rates of beta-blocker treatment (57.1% vs. 95.8%; p =0.003). The 30-day mortality was significantly higher (42.9% vs. 6.8%; p = 0.01) [39].

Lou B et al (2019) identified the clinical characteristics and protective factors of free wall rupture after myocardial infarction. Among the 5946 patients with AMI, 23 patients had a diagnosis of MFWR .18 (78.3%) patients were diagnosed with acute ST segment elevated myocardial infarction and the remaining 5 (21.7%) had acute non-ST segment elevated myocardial infarction. Early-phase MFWR happened in 12 (52.2%) and late-phase accounted for 8 (34.8%) in total. Late-phase MFWR had lower left ventricle ejection fraction value (45.8%±5.6% vs 63.0±3.8%, p<0.001) as compared with early-phase. Patients who survived from MFWR has higher ACE inhibitor/angiotensin II receptor blocker (ACEI/ARB) and β-blocker coverage in the in-hospital treatment of AMI (ACEI/ARB: 100.0% vs 35.3%, p=0.014; β-blocker: 100.0% vs 47.1%, p=0.048). They also found that reduced cardiac function corelates with higher incidence of later phase free wall rupture. Higher ACEI/ARB and β-blocker coverage in the AMI treatment strategy is associated with lower MFWR incidence [40].

Kageyama S et al (2020) examined the characteristics and mortality of VSR patients as well as the usefulness and appropriate timing of surgery. Among 3,947 consecutive patients with AMI 39 patients were diagnosed with VSR from 2002 to 2020. Patients' mean age was 76.3 years, and 33.3% of them were males. Most culprit lesions were located in the left anterior ascending artery (81.3%). The mean pulmonary-systemic flow ratio after VSR onset was 3.07 ± 1.98. On admission, 48.7% of patients were in a shock state. Surgical repair was possible in 28 patients at a median of 1 day after admission, with a mortality rate of 25%. Among all patients, the mortality rate was 43.6%. Survivors were significantly younger (71.3 vs. 82.7 years, p < 0.01), had higher mean arterial blood pressure (75.6 vs. 62.8 mmHg, p = 0.0496) and lower ejection fraction (44.3% vs. 54.8 %, p = 0.04), and underwent surgical repair more frequently (95.5% vs. 41.2%, p < 0.01) than the non-survivors. In multivariate analysis, younger age (p = 0.04) and surgical repair (p = 0.03) were significant predictors of survival. In surgical repair cases, time from admission to operation did not differ significantly

between survivors and non-survivors. Surgical repair and younger age were predictors of survival in patients with VSR, but the timing of surgery was not [41].

In a retrospective study Hao Z et al (2020) analysed incidence, risk factors and in-hospital outcomes related to cardiac rupture in 4190 ST-elevation myocardial infarction patients from 2004 to 2015 at Renji Hospital, School of Medicine, Shanghai Jiaotong University and reported that out of 4190 patients, 75 (1.8%) patients had cardiac rupture, including 33 at the ventricular septum and 42 at the left ventricle free wall (LVFW). Patients with cardiac rupture were more likely to be female, with more advanced age, lower rate of primary percutaneous coronary intervention (PPCI), and higher in-hospital mortality. Compared with survivors, the death cases were older, had a higher white blood cell count, higher rate of delayed admission (>12 h from symptom onset to door), earlier occurrence of cardiac rupture, higher percentage of LVFW rupture and lower rate of surgical repair. Logistic regression analysis showed that surgical repair served as the most valuable factor affecting survival. Moreover, elevated white blood cell count and advanced age might be related to an increased in-hospital death due to cardiac rupture [4].

In a study from eastern japan Yoneyama K et al (2021) conducted a retrospective analysis of multicenter registry data from eight medical universities and investigated the association between the time of cardiac rupture occurrence and the risk of in-hospital mortality after AMI. They included 183 patients from 10,278 patients who had cardiac rupture after AMI, and examined the incidence of in-hospital deaths during a median follow-up of 26 days. Patients were stratified into three groups according to the AMI-to-cardiac rupture time, namely the > 24-h group (n = 111), 24-48-h group (n = 20), and < 48-h group (n = 52). Around 87 (48%) patients experienced in-hospital death and 126 (67%) underwent a cardiac surgery. Multivariable Cox regression analysis revealed a non-linear association across the three groups for mortality (Hazard Ratio [CI]; < 24 h: 1.0, reference; 24-48 h: 0.73 [0.27-1.86]; > 48 h: 2.25 [1.22-4.15]) after adjustments for age, sex, Killip classification, percutaneous coronary intervention, blood pressure, creatinine, peak creatine kinase myocardial band fraction, left ventricular ejection fraction, and type of rupture. Cardiac surgery was independently associated with a reduction in the Hazard Ratio of mortality (HR [CI]: 0.27 [0.12-0.61])

and attenuated the association between the three AMI-to-cardiac rupture time categories and mortality (statistically non-significant) in the Cox model. They suggested that the AMI-to-cardiac rupture time contributes significantly to the risk of in-hospital mortality; however, rapid diagnosis and prompt surgical interventions are crucial for improving outcomes in patients with cardiac rupture after AMI [42].

In a retrospective analysis Hua K et al (2021) retrospective analysed 116 patients with post-infarction VSR hospitalised in Beijing Anzhen Hospital from January 2008 to February 2019 to assess long-term survival and identify risk factors for acute myocardial infarction in patients complicated with ventricular septal rupture (VSR). The overall in-hospital mortality rate was 47.4%. Logistic regression analysis revealed that age ($p<0.05$), female sex ($p<0.05$), no surgical repair ($p<0.05$), liver dysfunction ($p<0.05$), high heart rate ($p<0.05$), and low platelet count (PLT; $p<0.05$) were independent risk factors for in-hospital mortality. The 1-year mortality rate was lower in the surgery group than in the medical treatment group (18.3% vs 84.5%; $p<0.005$). During the mean follow-up of 5.2 ± 5.1 years (median, 1.3 years), the actuarial survival rates of these patients at 5 and 10 years were 72.3% and 43.2%, respectively. Overall in-hospital mortality rate remained high. The independent risk factors for in-hospital mortality associated with post-infarction VSR were age, female sex, no surgical repair, liver dysfunction, tachycardia, and low PLT level. The 1-year mortality and long-term outcomes of patients treated with surgery were significantly better than those of patients who were treated conservatively [43].

In a study from India J Kharge et al in 2022 studied the indicators and predictors of in-hospital mortality and survival in patients with ventricular septal rupture. Of total of 34,681 patients presenting with MI, 169 patients developed VSR. The incidence of VSR was 0.45%, survivors were mostly men and the mean age was 61.5 years; this was in contrast to the non-survivors, who were mainly women, and the mean age was 65.2 years ($p = 0.025$); had higher Killip Class (III-IV) ($p = 0.001$), lower LVEF ($p = 0.010$),had apical VSR and LV aneurysm ($p = 0.015$ and $p = 0.002$, respectively). 48 patients underwent CAG, with single vessel disease (SVD) with lower-grade Rentrop collateral flow being most common in the death group. 25 patients were subjected to definitive therapy with surgical patch closure or percutaneous device closure. The patients who died were older by

approximately 7 years. The risk factors for coronary artery disease, such as HTN, diabetes, and smoking, were not statistically different between the two groups [44].

MATERIAL AND METHODS

The study was conducted in department of cardiology at Dayanand Medical College and Hospital, Ludhiana (DMC&H).

Study Design: It was an observational study done in retrospective and prospective manner to study the clinical presentation and evaluate the management strategies and in-hospital outcome in patients of cardiac rupture.

SOURCE & METHOD OF COLLECTION OF DATA:

For retrospective analysis, data of all consecutive patients with cardiac rupture admitted in DMC&H during last 10 years was taken from institutional data base.

For prospective analysis, informed consent was taken from patient or patient's attendant presenting to emergency department of DMC&H with cardiac rupture from 1st August 2021 to 31st December 2022.

Inclusion Criterion:

- All patients of acute of myocardial infarction presenting to DMC&H with cardiac rupture on admission or diagnosed to have cardiac rupture during hospital stay.

Exclusion Criterion:

- Lack of informed consent.
- Patients with cardiac rupture from causes other than myocardial infarction.
- Patients with papillary muscle rupture were excluded.

The diagnosis of STEMI was based on typical clinical symptoms, electrocardiographic (ECG) findings of ST elevation .1 mV in more than two limb leads or .0.2 mV in two or more contiguous precordial leads, as well as documented cardiac enzyme (troponin I or T) elevation according to the definition of myocardial infarction from the Fourth Universal Definition of Myocardial Infarction.

Diagnosis of cardiac rupture: The occurrence of Ventricular septal rupture (VSR) was first suggested by physical examination findings such as cardiac systolic murmur. The diagnosis of Left ventricular free wall rupture (LVFWR) was made in cases either of sudden cardiogenic shock or low blood pressure associated with large pericardial effusion. Conventional or portable echocardiography were performed immediately once STEMI patients presented with new onset of systolic murmur, arterial hypotension or shock, or electrical mechanical dissociation in the absence of preceding heart failure. The clinical diagnosis of VSR was confirmed with color flow Doppler echocardiography.

Acute FWR was defined as an abrupt transmural rupture of the infarcted area, causing hemopericardium and death in <30 minutes.

Subacute FWR was defined as a gradual or incomplete rupture of the infarcted area with slow or recurrent bleeding into the pericardial sac, causing progressive or recurrent cardiac tamponade.

All patients who fulfills the criteria were evaluated and the data was collected in case record form. The database included data on the following elements: demographics for each patient (age, gender, past medical history), demographics for AMI on admission (clinical presentation Killip classification; blood pressure and heart rate; complete hemogram, serum creatinine, troponin-T, BNP, PCT, Total cholesterol, left ventricular ejection fraction; and coronary angiography data), data on cardiac rupture (septal rupture, free wall rupture, and day of rupture), and data on treatment (PCI/CABG and repaired surgery).

The time of STEMI onset was determined by a cardiologist as the time of occurrence of chest pain.

The time of cardiac rupture occurence was calculated by subtracting the time of cardiac rupture from the time of STEMI onset.

Treatment: All patients were given standard medication for AMI treatment, including aspirin, clopidogrel, low-molecular-weight heparin and statins. All other medications nitrates angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, beta blocker, antiarrhythmics and ionotropes were used as needed in patients. We used drug eluting stent during all PCI. Circulatory support with intra-aortic balloon counterpulsation (IABP) was considered after cardiac rupture was confirmed by echocardiography, with an urgent consultation on site being given by a cardiac surgeon. Generally, surgical repair was attempted in those hemodynamically stable with or without circulatory support of IABP. The management of cardiac rupture was at the discretion of the treating surgeon.

SURGICAL TECHNIQUE: The initial anesthetic and surgical protocol was essentially the same for all the patients. Left ventricular free wall rupture repair was achieved by 4-0 polypropylene sutures with supporting Teflon strips (direct suture technique) or by the autologous or synthetic pericardial patch and glue (patch-and-glue) technique. Ventricular septal rupture was repaired using double patch technique with 4/0 prolene. The left ventricle was then repaired with 3/0 prolene over a Teflon strips to buttress the repair. Concomitant CABG was not performed on all the patients but where feasible. Operations were performed on cardiopulmonary bypass with cardioplegic arrest.

OUTCOME: The patents were stratified into two groups: in-hospital survivors and non-survivors. The primary outcome was in-hospital mortality. Secondary outcome were factors associated with in-hospital mortality in patients of cardiac rupture.

STATISTICAL ANALYSIS: Data was described in terms of range; mean ± standard deviation (± SD), frequencies (number of cases) and

relative frequencies (percentages) as appropriate. To determine whether the data was normally distributed, a Kolmogorov-Smirnov test was used. Comparison of quantitative variables between the study groups was done using Student t-test and Mann Whitney U test for independent samples for parametric and non-parametric data respectively. For comparing categorical data, Chi square ($\chi 2$) test was performed and exact test was used when the expected frequency is less than 5. A probability value (p value) less than 0.05 was considered statistically significant. All statistical calculations were done using (Statistical Package for the Social Science) SPSS 21version (SPSS Inc., Chicago, IL, USA) statistical program for Microsoft Windows.

OBSERVATIONS & RESULTS

Table No. 1

Distribution of patients of cardiac rupture according to age

Age group (years)	Type of cardiac rupture				Total (n=82)
	Ventricular septal rupture (n=48)		Free wall rupture (n=34)		
< 50	1	2.1%	2	5.9%	3(3.7%)
50-60	13	27.1%	7	20.6%	20(24.4%)
60-70	25	52.1%	10	29.4%	35(42.7%)
70-80	7	14.6%	13	38.2%	20(24.4%)
> 80	2	4.2%	2	5.9%	4(4.9%)
Mean age (FWR)	67.03 ± 10.77 yrs				
Mean age (VSR)	64.73 ± 7.91 yrs				
Mean age (overall)	65.68 ± 9.21 yrs				

As shown in table, in the present study cardiac rupture was clinically confirmed in 82 patients. VSR was more common than FWR (58.5% VS 41.5%). The mean age for patients of FWR & VSR was (67.03 ± 10.77 & 64.73 ±7.91) yrs respectively. Overall, mean age for the study population was 65.68 ± 9.213 yrs. Overall, 3(3.7%) patients of cardiac rupture had age < 50 years, 20 (24.4%) patients were between 50-60 years, 35(42.7%) patients were between 60-70 years, 20(24.4%) patients were between 70-80 years, 4(4.9%) patients were > 80 years of age. On subgroup analysis the age distribution between the two groups was similar and majority of the study population in both the subgroups was older and above 60 years of age.

Figure No. 3

Distribution of patients of cardiac rupture according to age (years)

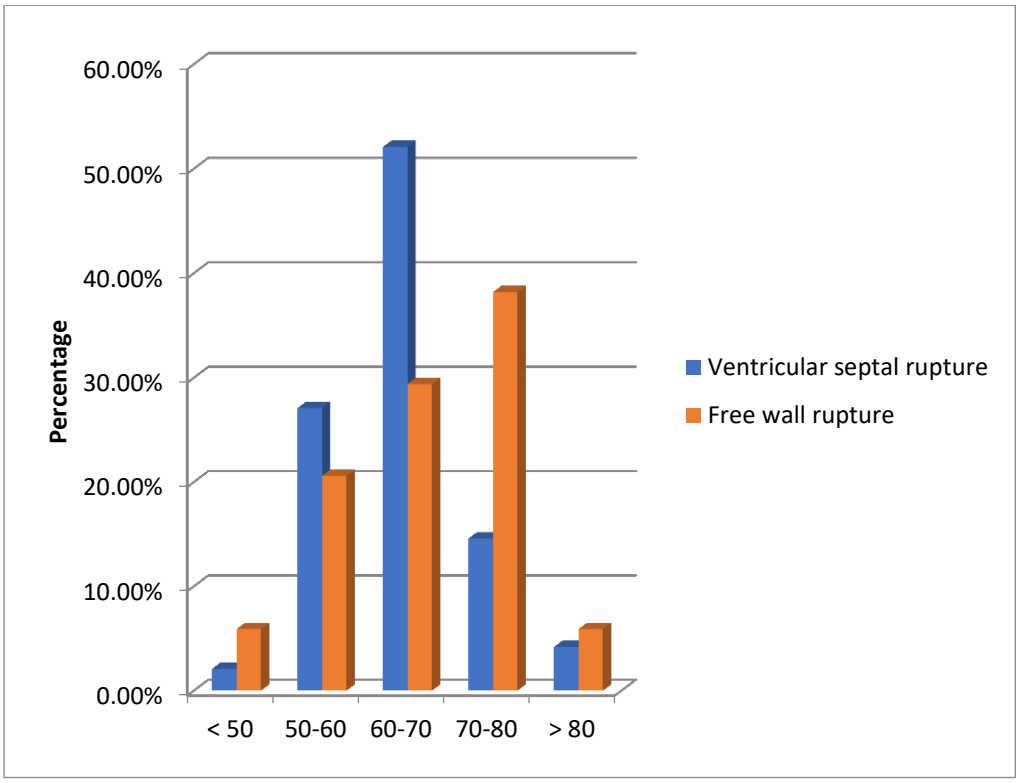

Table No. 2

Distribution of patients of cardiac rupture according to gender

Gender	Type of cardiac rupture				Total (n=82)
	Ventricular septal rupture (n=48)		Free wall rupture (n=34)		
Female	23	47.9%	14	41.2%	37(45.1%)
Male	25	52.1%	20	58.8%	45(54.9%)
Mean age (female)	66.78 ± 7.772 yrs				
Mean age (male)	64.78 ± 10.247 yrs				

As shown in table, in the present study 37(45.1%) patients were females while 45 (54.9%) patients were males. The mean age for female patients was 66.78 ± 7.772 yrs. The mean age for male patients was 64.78 ± 10.247yrs. On subgroup analysis the gender distribution between the groups was similar.

Figure No. 4

Distribution of patients of cardiac rupture according to gender

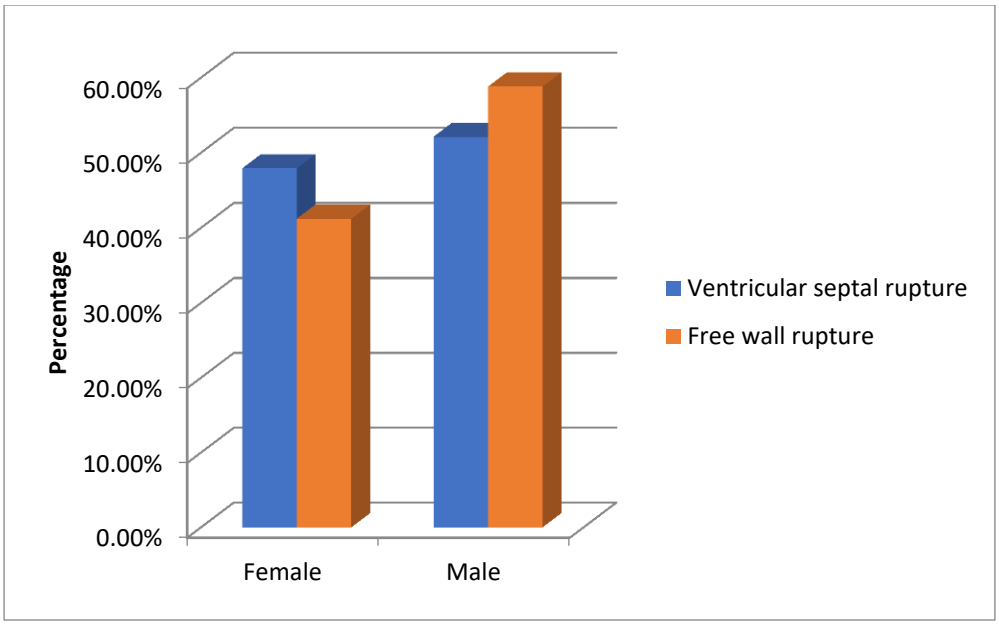

Table No. 3

Distribution of patients according to time from symptom onset to hospitalisation (hours)

Time from symptoms onset to hospitalisation (Hours)	Type of cardiac rupture				Total (n=82)
	Ventricular septal rupture (n=48)		Free wall rupture (n=34)		
0-12	6	12.5%	18	52.9%	24
12-24	4	8.3%	3	8.8%	7
24-48	12	25.0%	5	14.7%	17
48-72	9	18.8%	3	8.8%	12
72-96	10	20.8%	2	5.9%	12
96-120	5	10.4%	1	2.9%	6
> 120	2	4.2%	2	5.9%	4
Mean time (FWR)	29.38 ± 42.42 hrs				
Mean time (VSR)	54.94 ± 48.40 hrs				
Mean time (overall)	44.34 ± 47.46 hrs				

As shown in table, in the present study time from symptom onset to hospitalisation ranges from 1hr to 240 hrs. The patients of FWR had shorter mean time from symptom onset to hospitalisation when compared to patients of VSR (29.38 ± 42.42 vs 54.94 ± 48.40) hrs respectively. Overall mean time from symptom onset to hospitalisation for the study population was 44.34 ± 47.46 hrs. Delay in hospitalisation was more common in VSR subgroup. On comparing 42(87.5%) patients in VSR subgroups had delayed admission vs 16(47.1%)

patients in FWR subgroup. Overall, 58 (70.8%) patients out of 82 patients arrived late at hospital after symptom onset.

Figure No. 5

Distribution of patients according to time from symptom onset to hospitalisation (hours)

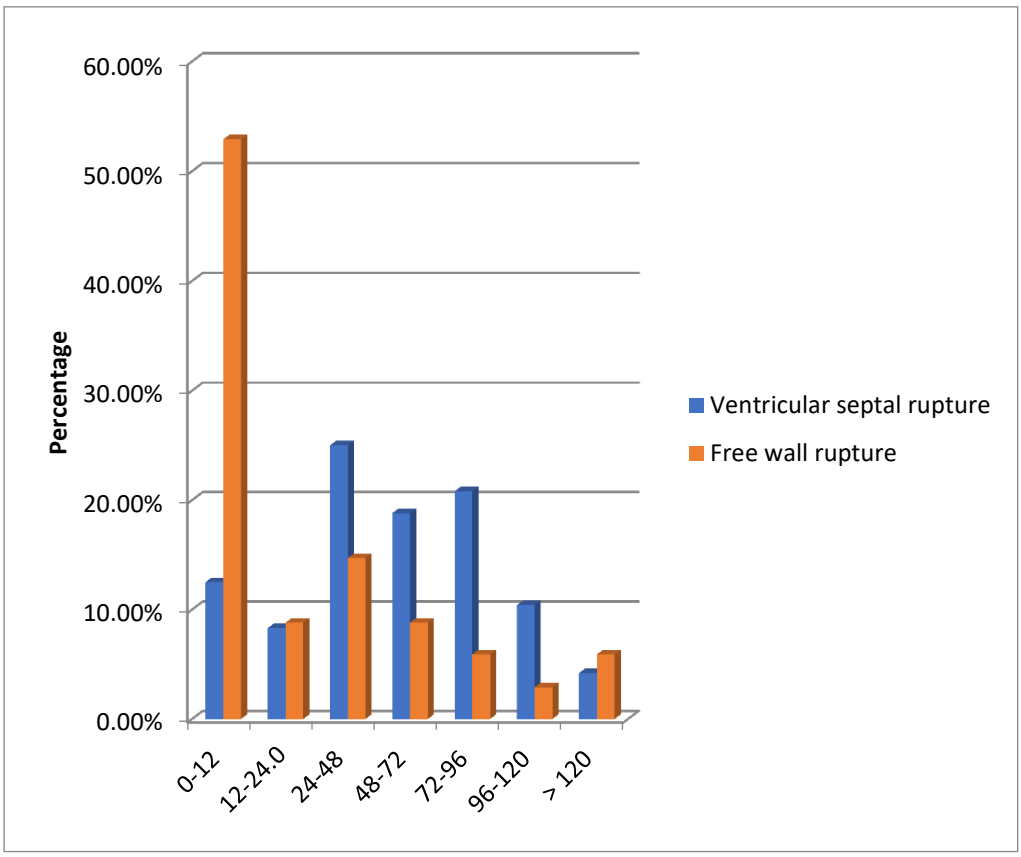

Table No. 4

Distribution of patients according to time of cardiac rupture occurrence after STEMI symptom onset (Days)

Time of Cardiac rupture occurrence after STEMI symptom onset (Days)	Type of cardiac rupture				Total (n=82)
	Ventricular septal rupture (n=48)		Free wall rupture (n=34)		
1	7	14.6%	15	44.1%	22(26.8%)
2	12	25.0%	7	20.6%	19(23.2%)
3	10	20.8%	3	8.8%	13(15.9%)
4	9	18.8%	4	11.8%	13(15.9%)
5	8	16.7%	3	8.8%	11(13.4%)
10	0	0.0%	1	2.9%	1(1.2%)
11	1	2.1%	0	0.0%	1(1.2%)
12	1	2.1%	0	0.0%	1(1.2%)
14	0	0.0%	1	2.9%	1(1.2%)
Mean time (FWR)	2.74 ± 2.76 days				
Mean time (VSR)	3.33 ± 2.17 days				
Mean time (overall)	3.09 ± 2.43 days				

As shown in table, in the present study the occurrence time of cardiac rupture ranged from 1 - 14 days after symptom onset of STEMI. The mean time of cardiac rupture from symptom onset in patients of FWR and VSR was (2.74 ± 2.76 vs 3.33 ± 2.17) days respectively. The overall mean time of cardiac rupture occurrence for study population

was 3.09 ± 2.43 days. On subgroups analysis majority of patients of FWR had early rupture (</=2) days after STEMI symptom onset (64.7% vs 39.6%) when compared to patients of VSR.

Figure No. 6

Distribution of patients according to time of cardiac rupture occurrence after STEMI symptom onset (Days)

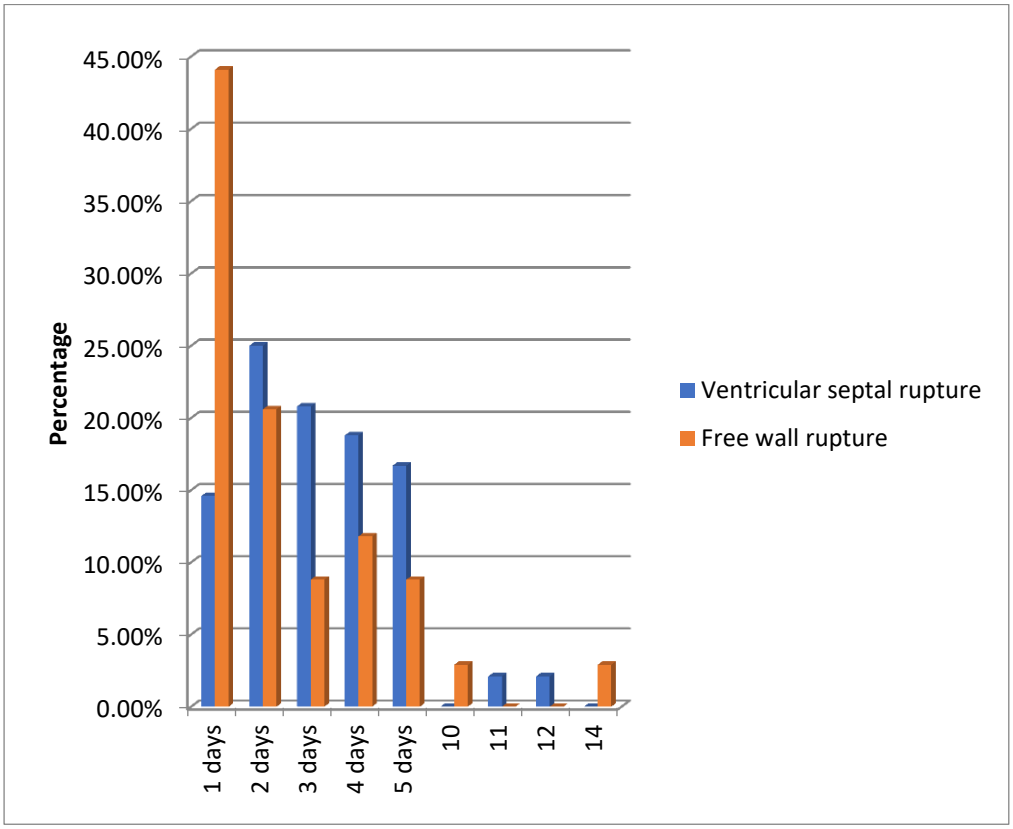

Table No.5

Distribution of co-morbidities among patients of cardiac rupture

Past medical history	Type of cardiac rupture				Total (n=82)
	Ventricular septal rupture (n=48)		Free wall rupture (n=34)		
Diabetes mellitus	26	54.2%	10	29.4%	36(43.9%)
Hypertension	30	62.5%	23	67.6%	53(64.6%)
Hypercholesterolemia	8	16.7%	11	32.4%	19(23.2%)
Smoking	8	16.7%	7	20.6%	15(18.3%)
CAD	2	4.2%	0	0.0%	2(2.4%)
PCI	1	2.1%	0	0.0%	1(1.2%)
CABG	1	2.1%	0	0.0%	1(1.2%)
Stroke /TIA	1	2.1%	1	2.9%	2(2.4%)
PAD	2	4.2%	0	0.0%	2(2.4%)
CKD	7	14.6%	2	5.9%	9(11.0%)
COPD	2	4.2%	4	11.8%	6(7.3%)

As shown in table, in the present study hypertension 53(64.6%) was the most common co-morbidity among patients of cardiac rupture followed by diabetes 36(43.9%), hypercholesterolemia 19(23.2%), smoking 15 (18.3%). Majority of patients 80(97.6%) didn't had any prior history of coronary artery disease. 1(1.2%) patient had prior history of PCI and CABG each. 2(2.4%) patients had prior history of CVA and PAD. 9(11.0%) patients had prior history of CKD. While 6(7.3%) patients had prior history of COPD.

Table No. 6

Clinical presentation at time of hospitalisation among patients of cardiac rupture

Clinical presentation at time of hospitalisation	Type of cardiac rupture				Total (n=82)
	Ventricular septal rupture (n=48)		Free wall rupture (n=34)		
Angina	14	29.2%	19	55.9%	33 (40.2%)
Cardiogenic shock	33	68.8%	12	35.3%	45 (54.9%)
Cardiac Arrest	1	2.1%	3	8.8%	4 (4.9%)

As shown in table, in the present study out of 82 patients of cardiac rupture 33(40.2%) patients presented with complaint of angina, 4(4.9%) patients presented in cardiac arrest, while 45 (54.9%) patients presented in cardiogenic shock at the time of hospitalisation. On subgroup analysis cardiogenic shock (68.8%) was most common clinical presentation in VSR patients, while angina (55.9%) was most common in FWR patients.

Table No. 7

Distribution of patients according to reperfusion therapy administered at time of admission.

Reperfusion therapy	Type of cardiac rupture				Total (n=82)
	Ventricular septal rupture (n=48)		Free wall rupture (n=34)		
None	38	79.2%	21	61.8%	59(72.0%)
Thrombolysis	9	18.8%	9	26.5%	18(22.0%)
PCI	1	2.1%	4	11.8%	5(6.1%)

As shown in table, in the present study only 5 (6.1%) patients received PCI out of which 4 patients received PPCI, 18 (22%) patients were thrombolysed while majority 59 (72%) of the patients of cardiac rupture were those patients who didn't received any form of reperfusion therapy.

Figure No. 7

Distribution of patients according to reperfusion therapy administered at time of admission.

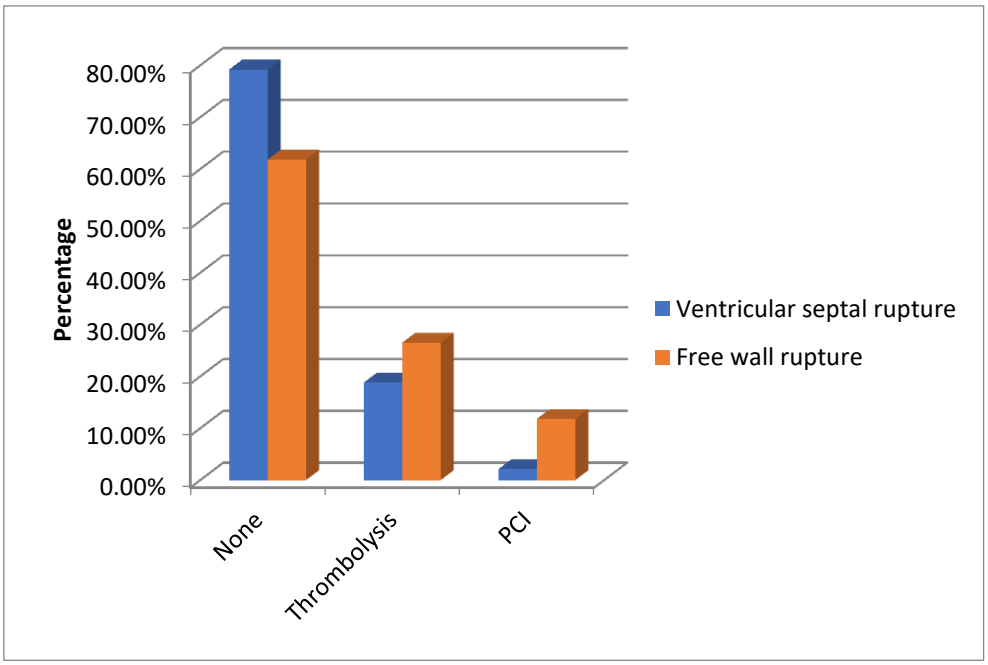

Table No. 8

Myocardial infarction site among patients of cardiac rupture

Myocardial Infarction Site	Type of cardiac rupture				Total (n=82)
	Ventricular septal rupture (n=48)		Free wall rupture (n=34)		
Inferior	8	16.7%	11	32.4%	19(23.2%)
Anterior	25	52.1%	9	26.5%	34(41.5%)
Multiple wall infarction (>2 walls)	15	31.3%	14	41.2%	29(35.4%)

As shown in table, in the present study among the patients of cardiac rupture, most common infarction site was anterior wall (41.5%) followed by inferior wall (23.2%). 29 (35.4%) patients had multiple wall(>2 wall) infarction. However, on subgroup analysis inferior wall infarction was more common than anterior wall infarction (32.4% vs 26.5%) among patients of FWR.

Figure No. 8

Myocardial infarction site among patients of cardiac rupture

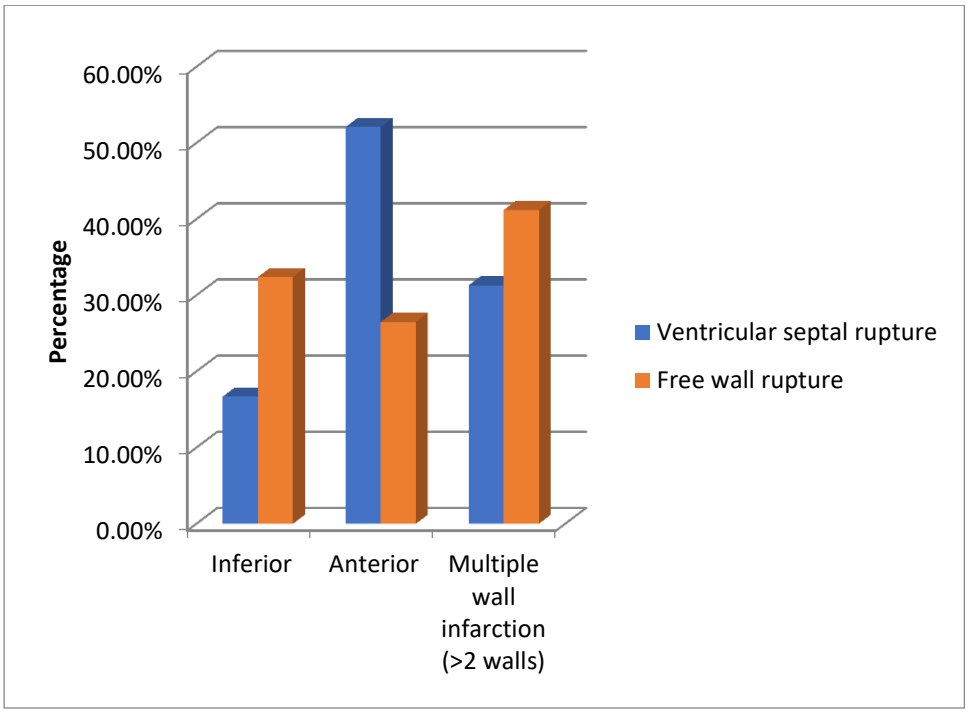

Table No. 9

Distribution of patients according to type & location of cardiac rupture

Type of Cardiac Rupture	No. of Patients	Percentage
Free wall rupture (FWR)	34	41.5%
Acute	13	38.2%
Sub-Acute	21	61.8%
Ventricular septal rupture (VSR)	48	58.5%
Apical Septum	33	68.7%
Basal Septum	13	27.1%
Mid septum	2	4.2%

As shown in table, in the present study, cardiac rupture was clinically confirmed in 82 patients. VSR was more common then LVFWR (58.5% VS 41.5%). Among VSR, apical septal rupture was the most common seen in 33(68.7%) patients followed by basal septum rupture seen in 13(27.1%) patients,2 (4.2%) patients had mid septum rupture. Among patients of LVFWR, acute rupture was seen in 13(38.2%) patients, while 21(61.8%) patients developed subacute rupture.

Figure No. 9a

Distribution of patients according to type of cardiac rupture

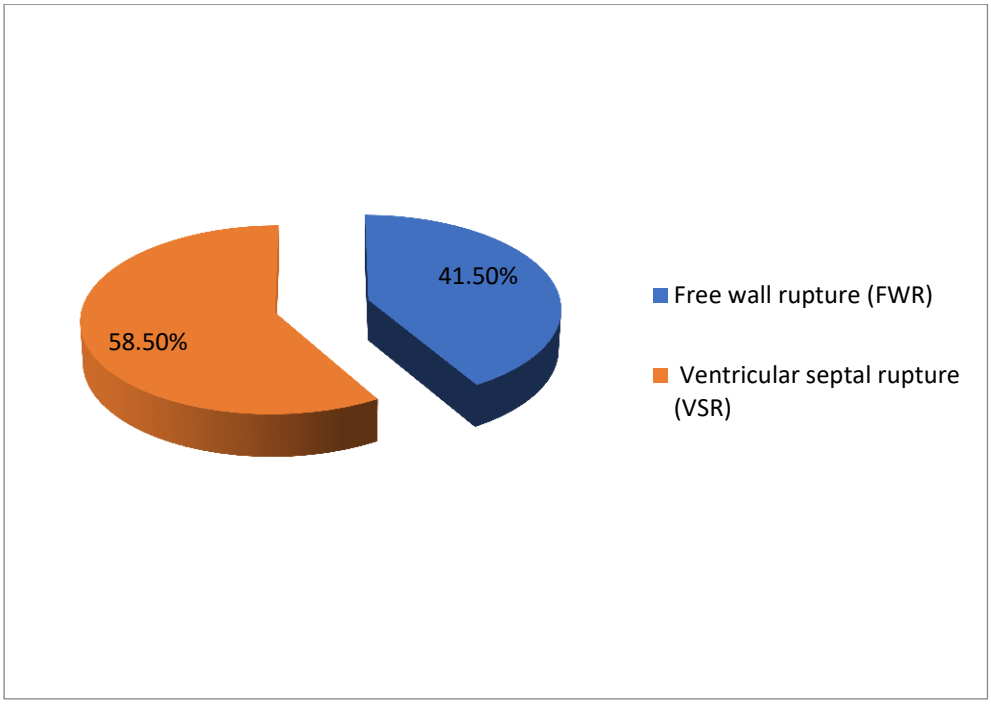

Figure No. 9b

Distribution of patients according to type & location of cardiac rupture

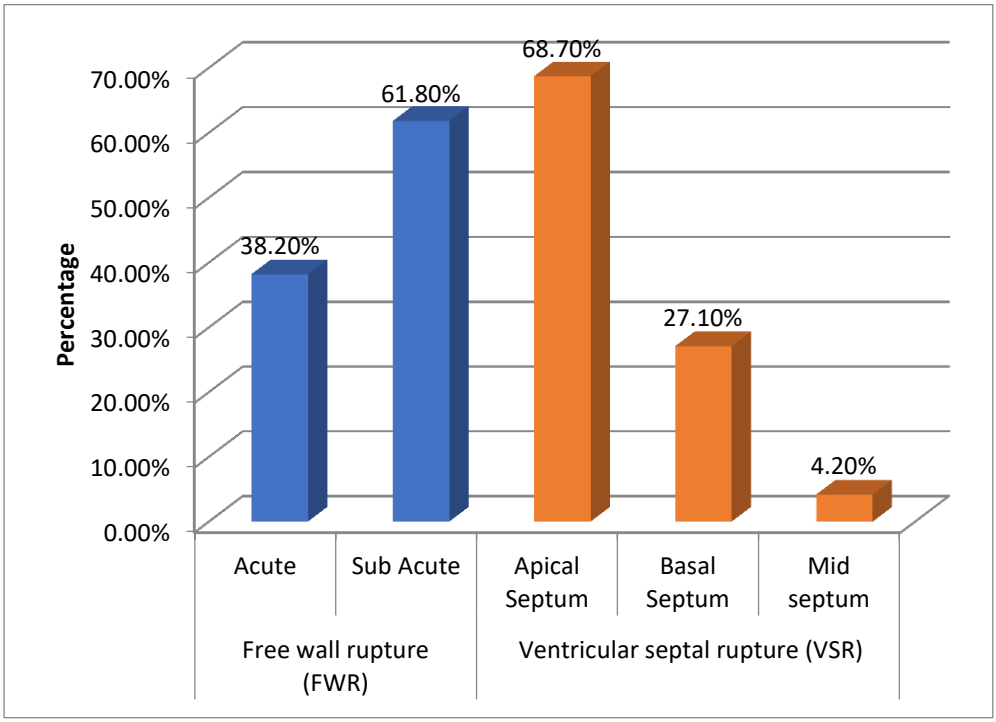

Table No.10

Coronary Angiography findings among patients of cardiac rupture.

Coronary angiography	Type of cardiac rupture				Total (n=30)
	Ventricular septal rupture (n=19)		Free wall rupture (n=11)		
SVD	10	52.6%	4	36.4%	14(46.7%)
DVD	6	31.6%	3	27.3%	9(30%)
TVD	3	15.8%	4	36.4%	7(23.3%)

As shown in table, in the present study, out of 82 patients cardiac rupture coronary angiography was performed in only 30(36.6%) patients. 19(39.5%) patients with VSR and 11(32.3%) patients with FWR underwent coronary angiography. 14(46.7%) patients had SVD, 9(30%) patients had DVD, and 7(23.3%) patients had TVD. On subgroup analysis angiographic findings were similar between the subgroups.

Table No. 11

Culprit vessel among patients of cardiac rupture.

Culprit Vessel	Type of cardiac rupture				Total (n=30)
	Ventricular septal rupture (n=19)		Free wall rupture (n=11)		
LAD	12	63.2%	6	54.6%	18(60%)
RCA	5	26.3%	1	9%	6(20%)
LCX	2	10.5%	4	36.4%	6(20%)

As shown in table, in the present study LAD was the most common culprit vessel among patients of cardiac rupture seen in 18(60%) patients, followed by RCA AND LCX in 6(20%) patients each. On subgroup analysis LAD was most common culprit vessel in both subgroups.

Table No.12

Baseline Investigations among patients of cardiac rupture

Baseline Investigations	Free wall rupture (n=34)		Ventricular septal rupture (n=48)		Total (n=82)	
	Mean	SD	Mean	SD	Mean	SD
Systolic BP (mmHg)	114.14	37.20	90.86	16.83	100.17	29.014
Diastolic BP (mmHg)	68.68	16.04	59.05	7.33	62.90	12.455
Hemoglobin (gm/dl)	12.76	2.08	12.03	1.80	12.33	1.941
TLC (10^3/ul)	14.20	4.18	16.15	5.83	15.34	5.273
Platelets (10^3/ul)	235.24	78.91	245.65	96.15	241.33	89.038
Creatinine (mg/dl)	1.15	0.33	1.77	1.14	1.51	0.949
Troponin-T (ng/ml)	3.36	2.41	2.78	1.90	3.02	2.131
BNP (pg/ml)	237.03	171.62	1516.88	818.68	986.21	896.311
PCT (ng/ml)	0.27	0.78	1.07	2.85	0.74	2.263
Cholesterol (mg/dl)	165.56	48.49	158.23	39.76	161.27	43.453
Ejection Fraction (E.F%)	34.44	5.25	32.52	5.07	33.32	5.204

As shown in table, in the present study patients of cardiac rupture have lower blood pressures, the mean Systolic and Diastolic BP were 100.17 ± 29.014 & 62.90 ± 12.455 (mmHg). Patients with cardiac rupture had elevated total leucocyte count (TLC) mean value was 15.34 ± 5.273(10^3/ul), higher creatinine mean creatinine was 1.51 ± 0.949 (mg/dl), elevated cardiac biomarkers mean troponin-T was 3.02 ± 2.131(ng/ml), & elevated natriuretic peptide levels mean BNP was 986.21 ± 896.311(pg/ml) & lower LVEF mean ejection fraction was 33.32 ± 5.204 % respectively. On comparing subgroups patients of VSR had lower mean systolic and diastolic blood pressures & higher mean serum creatinine & BNP levels than FWR patients.

Table No.13

Medication administered on admission in patients of cardiac rupture

Medication on Admission	Type of cardiac rupture				Total (n=82)
	Ventricular septal rupture (n=48)		Free wall rupture (n=34)		
Aspirin	48	100.0%	23	67.6%	71(86.6%)
P2Y12 inhibitor	25	52.1%	17	50.0%	42(51.2%)
Beta Blocker	0	0.0%	5	14.7%	5(6.1%)
ACE-Inhibitor	0	0.0%	5	14.7%	5(6.1%)
LMWH	46	95.8%	21	61.8%	67(81.7%)
Diuretics	20	41.7%	5	14.7%	25(30.5%)
Statins	46	95.8%	28	82.4%	74(90.2%)
Amiodarone	6	12.5%	1	2.9%	7(8.5%)
Ionotropes	45	93.8%	19	55.9%	64(78.0%)

As shown in table, in the present study out of 82 patients 71(86.6%) patients received aspirin, 42(51.2%) patients were given p2y12 inhibitor (clopidogrel mostly), 74 (90.2%) patients received statins, 67(81.7%) patients got LMWH .64(78%) patients required ionotropic support, 25(30.5%) patients needed diuretics, 7(8.5%) patients required amiodarone . Only 5(6.1%) patients were given Beta-Blocker and ACE-inhibitors at the time of hospital admission.

Table No.14

Treatment distribution among patients of cardiac rupture

Treatment	Type of cardiac rupture				Total (n=82)
	Ventricular septal rupture (n=48)		Free wall rupture (n=34)		
Pericardiocentesis	0	0.0%	12	35.3%	12(14.6%)
IABP	40	83.3%	6	17.6%	46(56.1%)
Concomitant CABG	4	8.3%	1	2.9%	5(6.1%)
Surgical repair	18	37.5%	5	14.7%	23(28%)
Conservative Management	30	62.5%	29	85.2%	59(72%)

As shown in table, in the present study out of 82 patients with cardiac rupture, 46(56.1%) patients were given mechanical circulatory support in the form of IABP (83.3% in VSR vs 17.6% in FWR) subgroup. Majority 59 (72%) patients were managed conservatively (62.5% in VSR vs 85.2% FWR) subgroup. Only 23(28%) patients were taken for surgical repair (37.5% in VSR vs 14.7% in FWR) subgroup. 5 (6.1%) patients underwent concomitant CABG (8.3% in VSR vs 2.9% in FWR) subgroup along with surgical repair. Pericardiocentesis was performed in 12(35.3%) patients of FWR.

Figure No. 10

Treatment distribution among patients of cardiac rupture

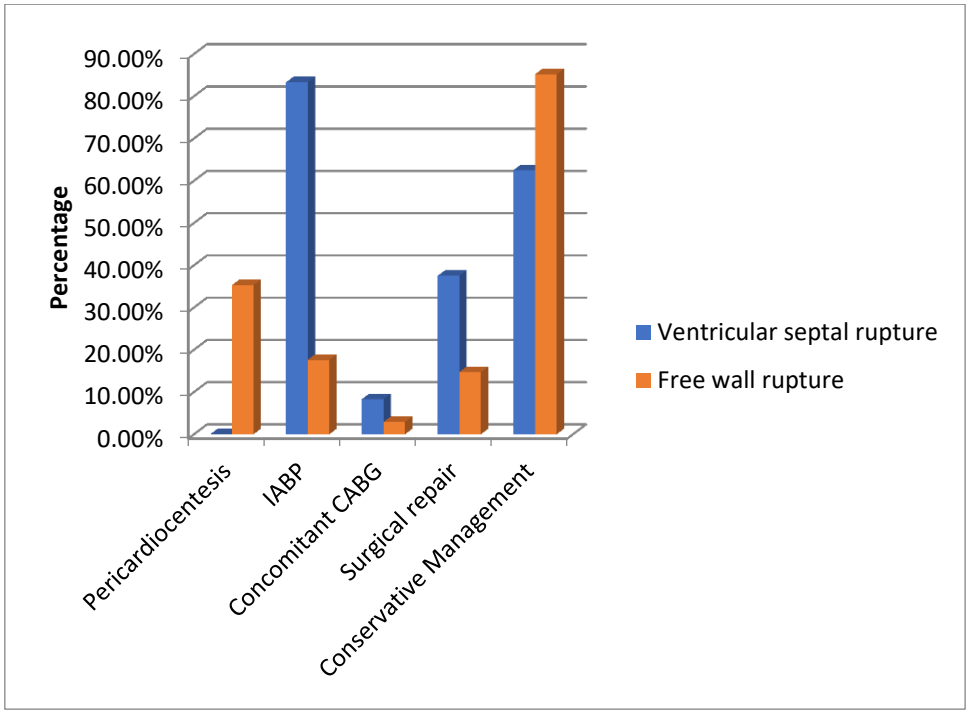

Table No.15

Time distribution from day of hospitalisation to surgical repair among patients of cardiac rupture

Surgical repair day from day of hospitalisation	Ventricular septal rupture (n=18)	Free wall rupture (n=5)
1	-	4
2	1	-
3	-	-
4	2	1
5	3	-
6	5	-
7	3	-
8	1	-
9	2	-
10	1	-
Mean time (FWR)	1.6 days	
Mean time (VSR)	6.77 days	

Table no,15 shows time distribution from day of hospitalisation to surgical repair among patients of cardiac rupture. Overall, out of 82 patients 23(28%) patients were managed with surgical repair. The mean time of surgical repair among patients of VSR from day of admission was 6.77 days. The mean time of surgical repair among patients of FWR from day of admission was 1.6 days.

Table No. 16

In- hospital Outcome in patients of cardiac rupture

OUTCOME	Type of cardiac rupture				Total (n=82)
	Ventricular septal rupture (n=48)		Free wall rupture (n=34)		
Discharge	16	33.3%	11	32.4%	27(32.9%)
Death	32	66.7%	23	67.6%	55(67.1%)

As shown in table, prognosis in both the subgroup of patients remains poor. The mortality in ventricular septal rupture patients was 66.7% whereas mortality in free wall rupture patients was 67.6%. The overall in- hospital mortality in patients of cardiac rupture was 67.1%.

Figure No. 11

In- hospital Outcome in patients of cardiac rupture

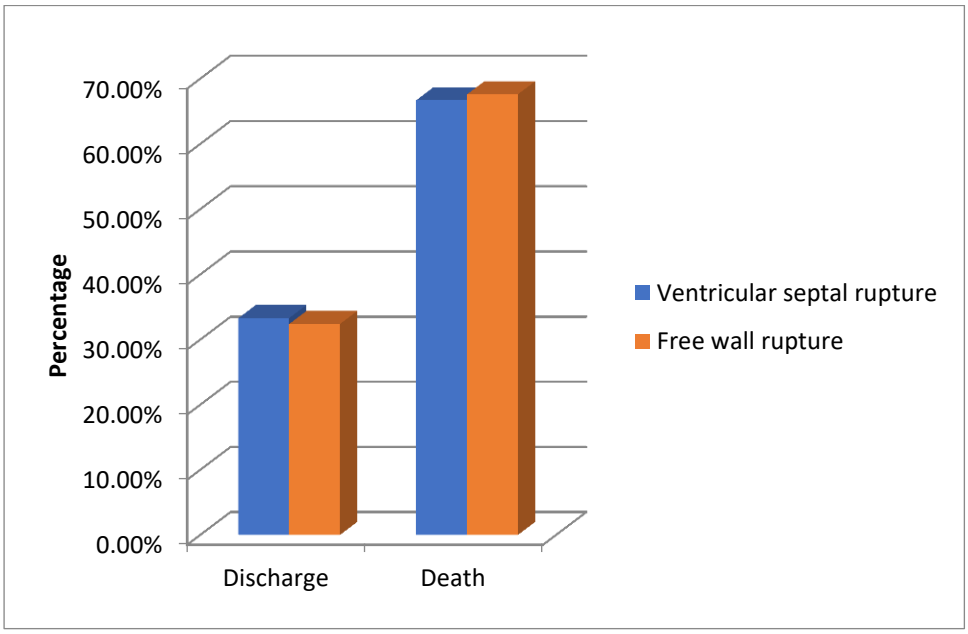

Table No.17

In-hospital Outcome in patients of free wall rupture

Management strategy	Survivors		Non-survivors		Total
	No. of cases	%age	No. of cases	%age	
Conservative	6	20.7%	23	79.3%	29(85.2%)
Surgical repair	5	100%	0	0.0%	5(14.8%)
Total	11	32.3%	23	67.6%	34

As shown in table, in the present study out of 34 patients of FWR 29(85.2%) patients were conservatively managed, while 5 (14.8%) patients were managed with surgical repair. Out of 29 patients managed conservatively 6(20.7%) survived while a majority 23(79.3%) patients died in the hospital. The in-hospital mortality for conservatively managed patients of FWR was 79.3%. 5 patients were managed surgically, all 5(100%) patients survived, none died during hospital stay. The in-hospital mortality in surgically managed patients was 0.0%. Overall, out of 34 patients 11(32.3%) patients survived, while 23(67.6%) patients died during hospital stay. The overall in-hospital mortality for the patients of FWR was 67.6%.

Figure No. 12

In-hospital Outcome in patients of free wall rupture

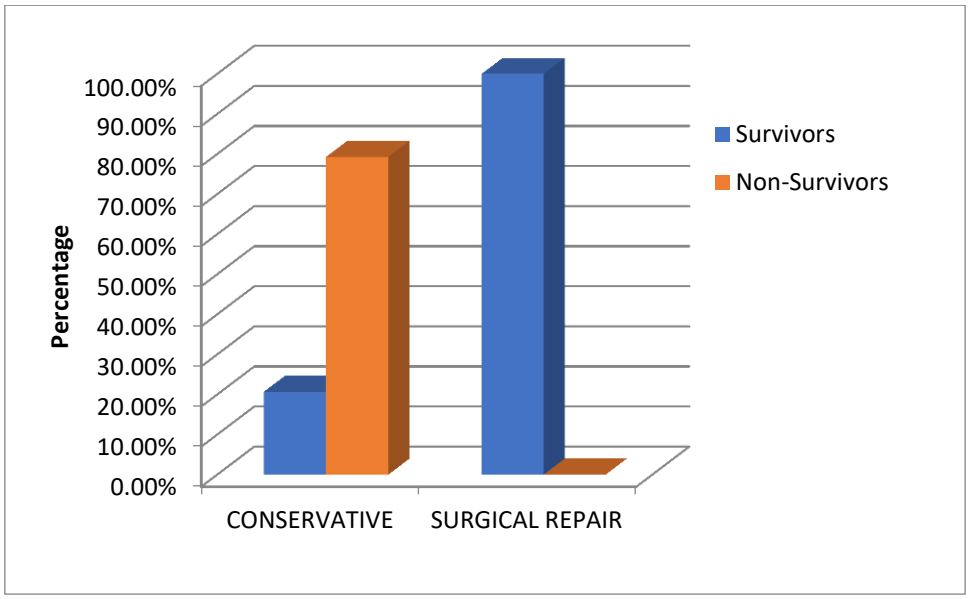

Table No.18

In-hospital Outcome in patients subacute free wall rupture

Management strategy	Survivors		Non-survivors		Total
	No. of cases	%age	No. of cases	%age	
Conservative	6	37.5%	10	62.5%	16(76.2%)
Surgical repair	5	100%	0	0.0%	5(23.8%)
Total	11	52.3%	10	47.7%	21

As shown in table, in the present study out of 21 patients of subacute FWR, 16 (76.2%) patients were conservatively managed, while 5 (14.8%) patients were managed with surgical repair. Out of 16 patients managed conservatively 6(37.5%) survived while 10(62.5%) patients died in the hospital. The in-hospital mortality for conservatively managed patients of subacute FWR was 62.5%. Out of 5 patients managed surgically all 5(100%) patients survived, none died during hospital stay. The in-hospital mortality in surgically managed patients of subacute FWR was 0.0%. Overall, out of 21 patients of subacute FWR 11(52.3%) patients survived, while 10(47.7%) patients died during hospital stay. The overall in-hospital mortality for the patients of subacute FWR was 47.7%.

Figure No. 13

In-hospital Outcome in patients subacute free wall rupture

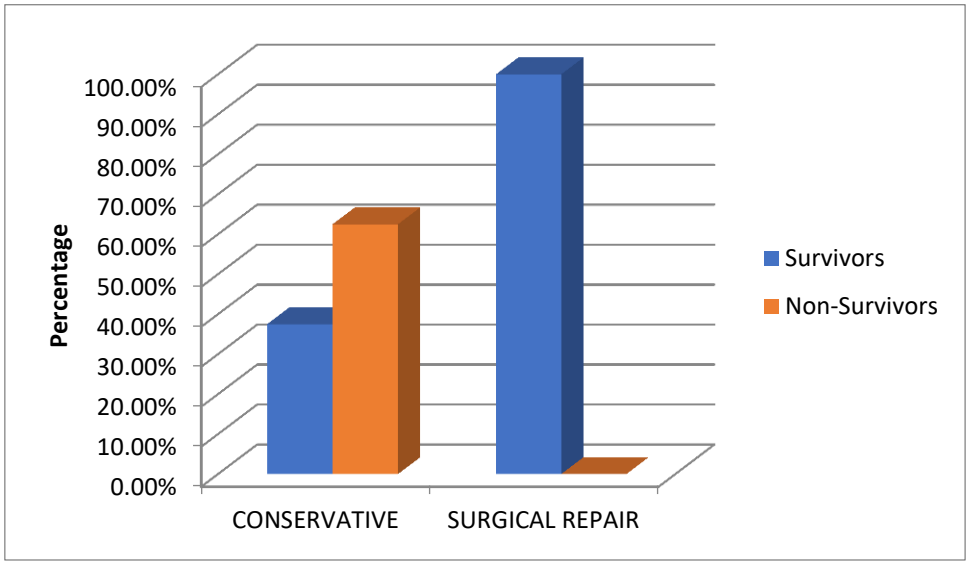

Table No.19

In-hospital Outcome in patients of Ventricular Septal Rupture

Ventricular septal rupture	Survivors		Non-survivors		Total
	No. of cases	%age	No. of cases	%age	
Conservative	1	3.4%	29	96.6%	30(62.5%)
Surgical repair	15	83.4%	3	16.6%	18(37.5%)
Total	16	33.3%	32	66.7%	48(100%)

As shown in table, in the present study out of 48 patients of Ventricular septal rupture 30 (62.5%) patients were conservatively managed, while 18 (37.5%) patients were managed with surgical repair. Out of 30 patients managed conservatively 1(3.4%) survived while a majority 29(96.6%) patients died in the hospital. The in-hospital mortality for conservatively managed patients of ventricular septal rupture was 96.6%. Out of 18 patients managed surgically 15(83.3%) patients survived, 3(16.6%) patient died in hospital. The in-hospital mortality in surgically managed patients of ventricular septal rupture was 16.6%. Overall, out of 48 patients 16 (33.3%) survived, while 32 (66.6%) patients died during hospital stay. The overall in-hospital mortality for the patients of Ventricular septal rupture was 66.6%.

Figure No. 14

In-hospital Outcome in patients of VSR

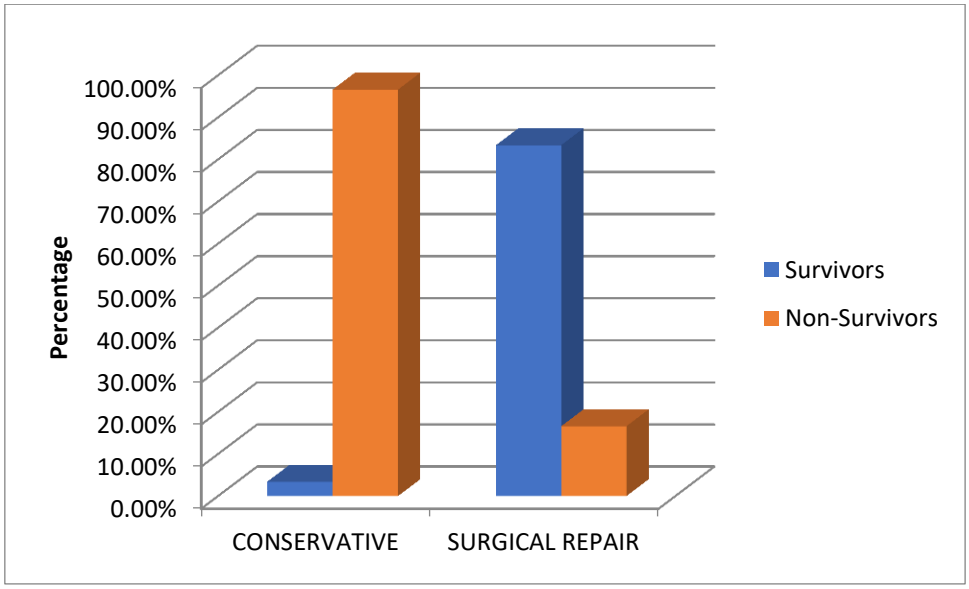

Table No. 20

Factors affecting In-hospital Outcomes in patients with cardiac rupture.

Events		Survivors (n=27)		Non-survivors (n=55)		Total	Chi-square value	p-value
		No. of cases	%age	No. of cases	%age			
Age group (years)	< 50	1	3.7%	2	3.6%	3	15.137	**0.004**
	50-60	11	40.7%	9	16.4%	20		
	60-70	14	51.9%	21	38.2%	35		
	70-80	0	0.0%	20	36.4%	20		
	> 80	1	3.7%	3	5.5%	4		
Gender	Female	9	33.3%	28	50.9%	37	2.259	0.133
	Male	18	66.7%	27	49.1%	45		
Past medical history	Diabetes mellitus	13	48.1%	23	41.8%	36	0.295	0.587
	Hypertension	14	51.9%	39	70.9%	53	2.877	0.09
	Hypercholesterolemia	7	25.9%	12	21.8%	19	0.172	0.679
	Smoking	7	25.9%	8	14.5%	15	1.569	0.21
	CAD	1	3.7%	1	1.8%	2	0.271	0.603
	PCI	1	3.7%	0	0.0%	1	2.062	0.151
	CABG	0	0.0%	1	1.8%	1	0.497	0.481
	Stroke/TIA	1	3.7%	1	1.8%	2	0.271	0.603
	PAD	0	0.0%	2	3.6%	2	1.006	0.316
	CKD	1	3.7%	8	14.5%	9	2.179	0.140
	COPD	2	7.4%	4	7.3%	6	0.010	0.982

As shown in table, in the present study patients of cardiac rupture were divided in two groups: survivor & non survivors. On univariate analysis, age >70yrs was significantly associated with in-hospital mortality (p value 0.004). However, the difference between the two groups was statistically insignificant (p value>0.05) on the basis of gender and Co-morbidities.

Table No 21

Factors affecting In-hospital Outcomes in patients with cardiac rupture

Events		Survivors (n=27)		Non-survivors (n=55)		Total	Chi-square value	p-value
		No. of cases	%age	No. of cases	%age			
Time from symptom onset to hospitalization (hours)	< 12	7	25.9%	17	30.9%	24	0.217	0.641
	> 12	20	74.1%	38	69.1%	58		
Time of cardiac rupture occurrence after STEMI symptom onset (days)	1	3	11.1%	19	34.5%	22	15.454	**0.004**
	2	3	11.1%	16	29.1%	19		
	3	5	18.5%	8	14.5%	13		
	4	9	33.3%	4	7.3%	13		
	> 5	7	25.9%	8	14.5%	15		
Clinical presentation at hospitalisation	Angina	13	48.1%	20	36.4%	33	1.046	0.306
	Cardiogenic shock	14	51.9%	31	56.4%	45	0.149	0.700
	Cardiac arrest	0	0.0%	4	7.3%	4	2.064	0.151
Killip class on admission	I	3	11.1%	10	18.2%	13	8.581	**0.035**
	II	5	18.5%	1	1.8%	6		
	III	5	18.5%	7	12.7%	12		
	IV	14	51.9%	37	67.3%	51		
Reperfusion therapy	None	19	70.4%	40	72.7%	59	0.05	0.823
	Thrombolysis	7	25.9%	11	20.0%	64	4.648	0.325
	PCI	1	3.7%	4	7.3%	5	0.403	0.526
Myocardial infarction site	Inferior	9	33.3%	10	18.2%	19	2.335	0.126
	Anterior	8	29.6%	26	47.3%	34	2.323	0.128
	Multiple walls (>2 walls)	10	37.0%	19	34.5%	29	0.049	0.824
Ejection Fraction (E.F%)	< 30	4	14.8%	14	25.5%	18	2.089	0.352
	30-40	21	77.8%	34	61.8%	55		
	40-50	2	7.4%	7	12.7%	9		

As shown in table, on univariate analysis cardiac rupture occurrence (</=2 days) and killip class IV were significantly associated with in-

hospital mortality with (p value 0.004 and 0.035) respectively. However, the difference between the two groups was statistically not significant on the basis of time from symptom onset to hospitalization, clinical presentation at hospitalization, reperfusion therapy administered on admission, myocardial infarction site and ejection fraction (p value>0.05).

Table No. 22

Factors affecting In-hospital Outcomes in patients with cardiac rupture

Baseline investigations	Survivors (n=27)		Non-survivors (n=55)		Z	p-value
	Mean	SD	Mean	SD		
Systolic BP (mmHg)	104.67	31.28	97.83	27.82	0.935	0.353
Diastolic BP (mmHg)	65.50	12.40	61.54	12.40	1.267	0.209
Heart Rate (/min)	103.19	16.19	100.78	24.06	0.469	0.640
Hemoglobin(gm/dl)	12.30	1.93	12.35	1.96	-0.111	0.912
TLC (10^3/ul)	13.33	4.24	16.32	5.48	-2.490	**0.015**
Platelets(10^3/ul)	244.74	109.80	239.65	77.96	0.242	0.810
Creatinine(mg/dl)	1.29	0.60	1.62	1.07	-1.527	0.131
Troponin-T(ng/ml)	1.66	1.12	3.69	2.20	-4.517	**0.001**
BNP (pg/ml)	917.22	1003.53	1020.07	846.54	-0.486	0.628
PCT (ng/ml)	0.36	0.61	0.92	2.72	-1.058	0.293
Total Cholesterol (mg/dl)	154.19	46.35	164.75	41.96	-1.035	0.304
Ejection Fraction (E.F%)	33.85	3.67	33.05	5.83	0.650	0.518

As shown in table, in patients of cardiac rupture elevated TLC and higher troponin-T were associated with in hospital mortality. The mean valve of TLC was 13.33 ± 4.24(10^3/ul) in survivors and 16.32 ± 5.48(10^3/ul) in non-survivors. On univariate analysis the difference between the two groups was statistically significant (p value=0.015). The mean valve of Troponin-T was1.66 ±1.12(ng/ml) in survivors and 3.69 ± 2.20 (ng/ml) in non-survivors. On univariate analysis, the difference between the two groups was statistically significant (p value = 0.001). However, the difference between the two groups was statistically insignificant (p value>0.05) on the basis of other parameters.

Table No. 23

Factors affecting In-hospital Outcomes in patients with cardiac rupture

Medication on Admission	Survivors (n=27)		Non-survivors (n=55)		Total	Chi-square value	p-value
	No. of cases	%age	No. of cases	%age			
Aspirin	22	81.5%	49	89.1%	71	0.903	0.342
P2Y12 inhibitor	12	44.4%	30	54.5%	42	0.740	0.390
Beta - Blocker	1	3.7%	4	7.3%	5	0.403	0.526
ACE-Inhibitor	2	7.4%	3	5.5%	5	0.121	0.728
LMWH	20	74.1%	47	85.5%	67	1.569	0.235
Diuretics	7	25.9%	18	32.7%	25	0.395	0.530
Statins	24	88.9%	50	90.9%	74	0.084	0.772
Amiodarone	2	7.4%	5	9.1%	7	0.066	0.798
Ionotropes	20	74.1%	44	80.0%	64	0.371	0.542

As shown in table, in the present study on univariate analysis, the difference between the two groups was statistically insignificant (p value>0.05) on the basis of medication given at the time of hospital admission in patients of cardiac rupture.

Table No. 24

Factors affecting In-hospital Outcomes in patients with cardiac rupture

Treatment	Survivors (n=27)		Non-survivors (n=55)		Total	Chi-square value	p-value
	No. of cases	%age	No. of cases	%age			
IABP	20	74.1%	26	47.3%	46	5.282	**0.032**
CABG	3	11.1%	2	3.6%	5	1.767	0.184
Conservative	7	25.9%	52	94.5%	59	42.253	**0.001**
Surgical repair	20	74.1%	3	5.5%	23		

As shown in table, in the present study on univariate analysis, use of mechanical circulatory support device i.e. IABP and surgical repair were associated with in hospital survival. The difference between the two groups was statistically significant with (p value=0.032 and 0.001) respectively.

Table No 25(a)

Factors affecting In-hospital Outcomes in patients with free wall rupture.

Events		OUTCOME				Total	Chi-square value	p-value
		Non-survivors (n=23)		Survivors (n=11)				
Age group (Years)	< 50	1	4.3%	1	9.1%	2	13.675	**0.008**
	50-60	2	8.7%	5	45.5%	7		
	60-70	5	21.7%	5	45.5%	10		
	70-80	13	56.5%	0	0.0%	13		
	> 80	2	8.7%	0	0.0%	2		
Gender	Female	13	56.5%	1	9.1%	14	6.911	**0.011**
	Male	10	43.5%	10	90.9%	20		
Time from symptom onset to hospitalization (Hours)	< 12	13	56.5%	5	45.5%	18	0.366	0.545
	> 12	10	43.5%	6	54.5%	16		
Clinical presentation at hospitalisation	Angina	14	60.9%	5	45.5%	19	0.717	0.397
	Cardiogenic shock	6	26.1%	6	54.5%	12	2.639	0.104
	Cardiac Arrest	3	13.0%	0	0.0%	3	1.574	0.535
Time of cardiac rupture occurrence after STEMI symptom onset (Days)	1	12	52.2%	3	27.3%	15	11.078	**0.026**
	2	7	30.4%	0	0.0%	7		
	3	1	4.3%	2	18.2%	3		
	4	1	4.3%	3	27.3%	4		
	> 5	2	8.7%	3	27.3%	5		
Reperfusion therapy	None	13	56.5%	8	72.7%	21	0.223	0.327
	Thrombolysis	6	26.1%	3	27.3%	9		
	PCI	4	17.4%	0	0.0%	4		
	Inferior	6	26.1%	5	45.5%	11	1.275	0.259

Myocardial infarction site	Anterior	8	34.8%	1	9.1%	9	2.523	0.112
	Multiple walls (>2 walls)	9	39.1%	5	45.5%	14	0.123	0.726
Ejection Fraction (E.F%)	< 30	3	13.0%	2	18.2%	5	2.815	0.245
	30-40	15	65.2%	9	81.8%	24		
	40-50	5	21.7%	0	0.0%	5		
TREATMENT	Pericardiocentesis	7	30.4%	5	45.5%	12	0.735	0.391
	IABP	1	4.3%	5	45.5%	6	8.652	**0.008**
	Concomitant CABG	0	0.0%	1	9.1%	1	2.154	0.142
	Surgical Repair	0	0.0%	5	45.5%	5	12.257	**0.001**
	Conservative	23	100%	6	54.5%	29		

Table No.25(b)

Factors affecting In-hospital Outcomes in patients with free wall rupture

Baseline Investigations	Non-survivors Death (n=23)		Survivors Discharge (n=11)		Z	p-value
	Mean	SD	Mean	SD		
Systolic BP (mmHg)	113.89	35.67	114.67	42.50	-0.050	0.960
Diastolic BP (mmHg)	67.63	16.09	70.89	16.65	-0.495	0.625
Heart Rate (b.pm)	91.09	22.17	107.55	20.78	-2.065	**0.047**
Hemoglobin (gm/dl)	12.53	2.28	13.25	1.55	-0.937	0.356
T.L.C (10^3/ul)	14.35	3.94	13.89	4.84	0.294	0.771
Platelets (10^3/ul)	243.04	78.31	218.91	81.41	0.830	0.413
Creatinine (mg/dl)	1.13	0.34	1.18	0.32	-0.422	0.676
Troponin- T (ng/ml)	4.00	2.60	2.02	1.19	2.397	**0.023**
BNP (pg/ml)	235.74	185.90	239.73	145.44	-0.062	0.951
PCT (ng/ml)	0.26	0.88	0.27	0.55	-0.043	0.966
Cholesterol (mg/dl)	168.83	43.20	158.73	59.80	0.562	0.578

On univariate analysis, factors associated with in-hospital survival in patients of FWR were:

- Use of IABP (p value =0.008)
- Surgical repair (p value =0.001)

On univariate analysis, factors associated with in-hospital mortality in patients of FWR were:

- Age > 70 yrs (p value =0.008)
- Female sex (p value =0.011)
- Early rupture (</=2 days) (p value =0.026).
- Lower heart rate (p value =0.047).
- Higher troponin-T (ng/ml) (p value =0.023)
- Conservative management (p value =0.001)

Table No. 26 (a)

Factors affecting In-hospital Outcomes in patients of Ventricular septal rupture

Events		OUTCOME				Total (n=48)	Chi-square value	p-value
		Non -Survivors (n=32)		Survivors (n=16)				
Age Group (Years)	< 50	1	3.1%	0	0.0%	1	5.292	0.259
	50-60	7	21.9%	6	37.5%	13		
	60-70	16	50.0%	9	56.3%	25		
	70-80	7	21.9%	0	0.0%	7		
	> 80	1	3.1%	1	6.3%	2		
Gender	Female	15	46.9%	8	50.0%	23	0.042	0.838
	Male	17	53.1%	8	50.0%	25		
Time from symptom onset to hospitalization (hours)	< 12	4	12.5%	2	12.5%	6		
	> 12	28	87.5%	14	87.5%	42	0.000	1.000
Clinical presentation at hospitalisation	Angina	6	18.8%	8	50.0%	14	5.042	**0.025**
	Cardiogenic shock	25	78.1%	8	50.0%	33	3.927	**0.048**
	Cardiac Arrest	1	3.1%	0	0.0%	1	0.511	0.475
Killip class	1	0	0.0%	1	6.3%	1	6.221	0.101
	2	1	3.1%	2	12.5%	3		
	3	5	15.6%	5	31.3%	10		
	4	26	81.3%	8	50.0%	34		
Time of cardiac rupture occurrence after STEMI symptom onset (days)	1	7	21.9%	0	0.0%	7	8.625	0.071
	2	9	28.1%	3	18.8%	12		
	3	7	21.9%	3	18.8%	10		
	4	3	9.4%	6	37.5%	9		
	> 5	6	18.8%	4	25.0%	10		
Reperfusion therapy	None	27	84.4%	11	68.8%	38	1.746	0.417
	Thrombolysis	5	15.6%	4	25.0%	9		
	PCI	0	0.0%	1	6.3%	1		
Myocardial infarction	Inferior	4	12.5%	4	25.0%	8	1.2	0.273
	Anterior	18	56.3%	7	43.8%	25	0.668	0.414

site	Multiple walls (>2 walls)	10	31.3%	5	31.3%	15	0.000	1.000
Ejection Fraction (E.F%)	< 30	11	34.4%	2	12.5%	13	2.788	0.248
	30-40	19	59.4%	12	75.0%	31		
	40-50	2	6.3%	2	12.5%	4		
Ventricular septal rupture	Apical Septum	22	68.8%	11	68.8%	33	0.288	0.866
	Basal Septum	9	28.1%	4	25.0%	13		
	Mid septum	1	3.1%	1	6.3%	2		
Treatment	IABP	25	78.1%	15	93.8%	40	1.875	0.171
	CABG	2	6.3%	2	12.5%	4	0.545	0.46
	Surgical repair	3	9.4%	15	93.8%	18	32.4	0.001
	Conservative	29	90.6%	1	6.2%	30		

Table No. 26(b)

Factors affecting In-hospital Outcomes in patients with Ventricular septal rupture

Baseline investigations	Non -Survivors (n=32)		Survivors (n=16)		Z	p-value
	Mean	SD	Mean	SD		
Systolic BP (mmHg)	86.52	11.72	98.67	21.75	-2.363	**0.023**
Diastolic BP (mmHg)	57.26	6.38	62.27	8.03	-2.221	**0.032**
Heart Rate	107.75	23.24	100.19	11.95	1.219	0.229
Hemoglobin (gm/dl)	12.22	1.73	11.64	1.93	1.039	0.304
T.L.C (10^3/ul)	17.74	6.03	12.95	3.90	2.886	**0.006**
Platelets (10^3/ul)	237.22	78.87	262.50	125.12	-0.856	0.396
Creatinine (mg/dl)	1.98	1.26	1.36	0.74	1.818	0.076
Troponin- T (ng/ml)	3.46	1.87	1.40	1.03	4.088	**0.000**
BNP (pg/ml)	1583.81	662.81	1383.00	1078.64	0.798	0.429
PCT (ng/ml)	1.39	3.43	0.42	0.66	1.123	0.267
Cholesterol(mg/dl)	161.81	41.49	151.06	36.24	0.881	0.383

On univariate analysis factors associated with in-hospital survival in patients of VSR were:

- Surgical repair (p value =0.001)

On univariate analysis factors associated with in-hospital mortality in patients of VSR were:

- Cardiogenic shock on presentation (p value =0.048)
- Low mean systolic and diastolic BP (p value =0.023 & 0.032)
- Elevated white cell count (TLC) (p value=0.006)
- Higher troponin-T (ng/ml) (p value =0.000)
- Conservative management (p value =0.001)

DISCUSSION

Despite significant evolution of the pathophysiological basis of cardiac rupture and improvement in diagnostic as well as therapeutic strategy, cardiac rupture continues to challenge medical and surgical fraternity to improve prognosis and reduce cardiac morbidity and mortality.

The incidence of ST-elevation myocardial infarction (STEMI) is much higher in the Indian subcontinent than Western population [45]. It is estimated that approximately 3 million cases of STEMI occur every year. According to the Position statement for the management of STEMI in India, delayed hospitalization and reperfusion therapy are causes of increased 30-day mortality [46-49].

Clinical characteristics of patients of cardiac rupture:

Of all the factors related to cardiac rupture, age is probably the most important. In present study, majority of the study population was older and above 60 years of age. Overall, mean age for the study population was 65.68 ± 9.213 yrs. Our findings are similar to previous studies who have reported that patients of cardiac rupture are elderly (commonly in-between 65 and 70 years) [50-53].

Previous studies have suggested that cardiac rupture is more common in female then males (although no definitive sex bias has been reported but relatively more common in females due to lower reported incidence of AMI in females) [6-8,54]. In our study, we noted a male preponderance, contrary to the traditional view of higher female predisposition, as 37(45.1%) patients were females while 45(54.9%) patients were males.

Previous studies have postulated hypertension to be of significance in the pathogenesis of cardiac rupture [54-57]. High blood dramatically increases intracavitary pressures and shear stress force against the necrotic area during myocardial contraction, leading to a tear [58]. In present study 53(64.6%) patients were known hypertensive.

Previous studies have reported that diabetes mellitus and peripheral vascular disease are less likely in patients of cardiac rupture, as these conditions are associated with the development of collateral circulation, which may reduce the possibility of a rupture [59]. In present study 36(43.9%) patients had diabetes & only 2(2.4%) patients had prior PAD. Our findings are in concordance with the previous studies.

Previous studies have reported that cardiac rupture occurs more frequently in first myocardial infarcts due to complete coronary artery occlusion without collateral circulation [60,61]. This hypothesis was further supported by Pretre et al. who described the development of VSR in a particularly vulnerable myocardium following an abrupt closure of a single vessel [61,62] following acute MI supporting the theory of absence of collaterals leading to VSR. Our findings are consistent with the findings of previous studies as 80(97.6%) patients in the present study didn't had any prior history of MI.

Ino et al [63] reported that single vessel disease, absence of primary PCI, are independent predictors of cardiac rupture. Our findings are consistent with these as 46.7% patients had SVD, only 4.8% patients of cardiac rupture underwent primary PCI.

During the era of thrombolytic therapy, or the no reperfusion era, the incidence of cardiac rupture after STEMI was 6% [1]. In recent years, the incidence of cardiac rupture after AMI has decreased to less than 2% with the widespread use of PPCI [3]. These findings indicate that reperfusion therapy especially PPCI, can prevent transmural progression of myocardial necrosis through early recanalization of the infarct-related artery [64] and patients who do not receive reperfusion therapy are at higher risk of cardiac rupture. Our findings are consistent with these observations as in the present study majority 59(72%) of the patients of cardiac rupture were those patients who didn't received any form of reperfusion therapy. 18 (22%) patients developed cardiac rupture after thrombolysis, while 5 (6.1%) patients developed cardiac rupture after PCI, out of which 4 patients underwent primary PCI.

Previous studies presented conflicting views site of cardiac rupture. Some studies reported that the anterior wall was more susceptible, other reported that rupture is more common on the lateral or posterior wall [1,21,59,65-66]. Despite rupture being theoretically more probable in the inferior lateral wall, it seems that anterior infarcts are more common [67]. However, a thorough literature review showed that, in specific cases of subacute LV wall rupture, inferior infarctions seem to be involved [7,14,20]. Our findings are consistent with this as in the present study, most common infarction site was anterior wall (41.5%) followed by inferior wall (23.2%). 29 (35.4%) patients had multiple wall (>2 walls) infarction. However, among patients of FWR inferior wall infarction was more common than anterior wall infarction (32.4% vs 26.5%).

In a recent study of patients with cardiac rupture, Okamura H et [68] reported the LAD was the most common culprit vessel seen in 46% of the cases. In present study also LAD was the most common culprit vessel seen in (60%) patients who were taken for angiography.

Previous studies have reported that cardiac rupture appears in the first week (usually on the fourth or fifth day) post-AMI, although this may vary from few minutes to sometimes more than a month after AMI[14,21,68-69]. In the present study, the occurrence time of cardiac rupture ranged from 1hr - 14 days after symptom onset of STEMI. (95.2%) of patients had cardiac rupture during first 5 days after symptom onset of STEMI. Only 2(2.4 %) patients experienced ruptures a week after AMI onset. Our findings are in concordance with the findings of previous studies.

The mean time of cardiac rupture occurrence was 3.09 ± 2.43 days. In a recent study from china by Hao Z et al [4] cardiac ruptures occurred within 4 h to 17 days from AMI onset, with a mean onset time of 4.4 days.

In present study 48(58.5%) patients had cardiac rupture during first 48 hrs after symptom onset, suggesting that cardiac rupture was more common with in first 48 hrs after AMI, and was less common after 48 hrs had lapsed. Yoneyama K et al [42] also made similar observations.

Factors associated with in-hospital mortality in patients of cardiac rupture

In present study we found that older age >70yrs was significantly associated with in-hospital mortality in patients of cardiac rupture (p value= 0.004). Gender of the patient had no significant association with the in-hospital mortality (p value = 0.133). Our findings are in concordance with the findings made by Hao Z et al [4]. However, on subgroup analysis among patients of FWR, females were found to have higher in-hospital mortality compared to males (p value0.011).

In the present study, past medical history (hypertension, diabetes, prior MI, etc), was not found to be associated with in-hospital mortality. (p value > 0.05). Kharge J et al [44] also reported similar findings in patients of post-MI VSR patients.

In the present study we found that early cardiac rupture (</=2 days) was significantly associated with in-hospital mortality (p value 0.004). Similar findings were observed by Nozoe M et al [36], Yoneyama K et al[42] who reported that early rupture has higher mortality then late rupture.

In the present study we found that delayed hospitalisation (>12h) was not associated with in hospital mortality (p value 0.641). Similar findings were observed by Formica et al [21] demonstrated that mortality in LVFWR patients was not affected by early (5–10 h) versus late (10 h) presentation. However, Hao Z et al [4] reported that delayed hospital admission was associated with higher in-hospital mortality in patients of cardiac rupture. (p value 0.0278).

In the present study we did not find any statistically significant correlation between initial clinical presentation and in-hospital outcome.

(p value > 0.05). However, Formica et al [21] reported that cardiac arrest on presentation was the most important predictor for in-hospital mortality in patients of LVFWR (p value < 0.0001).

In the present study, we found that Killip class IV on hospital admission was associated with in-hospital mortality (p value 0.035). Kharge J et al [44] also reported similar findings in patients of post-MI VSR.

Huang et al [37] in post-MI VSR reported that lower LVEF and cardiogenic shock were early predictors of early in-hospital mortality. In the present study cardiogenic shock on presentation was found to be associated with in-hospital mortality in post MI VSR patients (p value 0.048). However, we did not LVEF to be significantly associated with in-hospital mortality (p value 0.518).

In present study, we found that elevated TLC & higher troponin-T were associated with in-hospital mortality (p value 0.015 & 0.001) respectively. Hao Z et al [4] also reported similar findings in patients of cardiac rupture.

Lou B et al [40] reported that patients who survived from MFWR has higher ACE inhibitor/angiotensin II receptor blocker (ACEI/ARB) and β-blocker coverage in the in-hospital treatment of AMI (ACEI/ARB, p=0.014; β-blocker, p=0.048). However, in the present study we did not ACEI/ARB & β-blocker to be associated with in-hospital survival. Only 5(6.1%) patients in the present study were given ACEI/ARB & β-blocker.

Liebelt JJ et al [70], reported that main cause of death in AMI patients with cardiac rupture is “pump failure”, which results in hemodynamic instability. Therefore, it is urgently necessary to improve the haemodynamic status of patients to reduce early mortality. With the rapid advancement in machinery circulation technology, the survival rate of patients with AMI complicated by cardiac rupture has increased, especially when ECMO and IABP are used pre-operatively [70]. Pre-operative IABP will increase cardiac output, and improve coronary

perfusion until surgical repair is performed. In our study, on univariate analysis, we found the use of IABP pre-operatively was associated with survival in patients of cardiac rupture (p value 0.032). However, logistic multivariate analysis showed that the use of IABP was not the protective factor for in-hospital mortality. Hao Z et al [4] also reported similar findings in patients of cardiac rupture.

Bates ER [10] reported that the mortality rate for free-wall rupture is 75% to 90%, and the mortality rate for ventricular septal rupture is 40% to 75%. In the present study, the mortality in free wall rupture patients was 67.6%. whereas mortality in ventricular septal rupture patients was 66.7%. The overall in- hospital mortality in patients of cardiac rupture was 67.1%.

Our observations corroborate O'Rourke's [65] that acute FWR did not permit time for therapeutic intervention & results in death within a few minutes (<30 min). In this study, we found that acute LVFWR rupture was associated with 100% in-hospital mortality.

In present study, we found that acute LVFWR rupture is associated with a higher in-hospital mortality rate compared with subacute (100% vs 47.7%), Formica et al [21] also made similar observations

Due to the poor prognosis of conservative strategies [20], surgical repair is typically the cornerstone of the treatment strategy in patients of LVFWR. However previous studies [14] have reported that a small subset of patients of subacute FWR can be managed conservatively in whom a small tear might be temporarily sealed by a clot or fibrinous pericardial adhesions that may be compatible with life for several hours or even longer. In the present study out of 16 patients of subacute FWR managed conservatively 6 survived. Hence, we found that a small subset of subacute FWR patients can be managed conservatively. Our findings are in concordance with previous studies.

In present study we found that location of VSR was not associated with in-hospital mortality (p value 0.866). Kharge J et al [44] also reported similar findings in patients of post-MI VSR.

In present study only 5 (6.1%) patients underwent concomitant CABG so we could not demonstrate beneficial effect of concomitant CABG. However, we can postulate that the real effectiveness of the myocardial revascularization is underestimated by the small number of patients undergoing CABG. Furthermore, coronary angiography is not always possible in cardiac rupture patients, only 30(36.6%) out of 82 patients underwent coronary angiography in present study.

STRENGTHS & LIMITATIONS

Strength: The strength of study is that we examined multiple factors that contribute to mortality in patients of cardiac rupture. The results of our study would be very useful when clinicians are faced with a cardiac rupture. Our findings indicate that prompt diagnosis by use of bedside echocardiography and surgical intervention are important to improve outcomes in patients of cardiac rupture after AMI.

Limitations: We acknowledge limitations in present study. First ours is a single centre study using a small sample size. Secondly in present study we did not include patients with MI without cardiac rupture. So, we could not determine incidence of cardiac rupture & factors that would prevent rupture. Another limitation of our study is that we did not took into account the contribution of out of hospital deaths. So, we could have underestimated the true mortality in patients of cardiac rupture. Another limitation is that we did not consider door to ballon & door to needle time which are important factors in in-hospital mortality. Even though surgical management fared better than conservative it is to be noted that surgery was not performed in high risk patients. The decision was taken by treating surgeon. So, more multicentric studies with large no of patients are needed to confirm our findings.

SUMMARY & CONCLUSION

The present study was conducted in department of cardiology at Dayanand Medical College and Hospital, Ludhiana. It was an observational study done in retrospective and prospective manner to study the clinical presentation and evaluate the management strategies and in-hospital outcome in patients of cardiac rupture. 82 patients with clinical diagnosis of cardiac rupture admitted in DMC&H who fulfilled the inclusion and exclusion criteria, were taken up for the study. Patients were assessed on basis of history, physical examination, investigation and treatment received.

- In the present study, cardiac rupture was clinically confirmed in 82 patients. VSR was more common than FWR (58.5% VS 41.5%). The patients of FWR were older than patients of VSR. The difference between mean age was (67.03 ± 10.77 vs 64.73 ± 7.91) yrs respectively. Overall mean age for the study population was 65.68 ± 9.213 yrs.
- In the present study, 3(3.7%) patients of cardiac rupture had age < 50 years, 20 (24.4%) patients were between 50-60 years, 35(42.7%) patients were between 60-70 years, 20(24.4%) patients were between 70-80 years , 4(4.9%) patients were > 80 years of age.On subgroup analysis the age distribution between the two groups was similar and majority of the study population in both the subgroups was older and above 60 years of age.
- In the present study 37(45.1%) patients were females while 45(54.9%) patients were males. The mean age for female patients was 66.78 ± 7.772 yrs. The mean age for male patients was 64.78 ± 10.247 yrs.
- In the present study time from symptom onset to hospitalisation ranges from 1hr to 240 hrs. The overall mean time from symptom onset to hospitalisation for the study population was 44.34 ± 47.46 hrs. On subgroup analysis, we found that patients of FWR had significantly shorter mean time from symptom onset to

hospitalisation when compared to patients of VSR (29.38 ± 42.42 vs 54.94 ± 48.40) hrs respectively.

- Overall, 58 (70.8%) patients out of 82 patients arrived late (>12 hrs) at hospital after symptom onset. Delay in hospitalisation was more common in VSR subgroup. On comparing 42 (87.5%) patients in VSR subgroups had delayed admission vs 16 (47.1%) patients in FWR subgroup.
- In the present study the occurrence time of cardiac rupture ranged from 1 - 14 days after symptom onset of STEMI. The mean time of cardiac rupture from symptom onset in patients of FWR and VSR was (2.74 ± 2.76 & 3.33 ± 2.17) days respectively. The overall mean time of cardiac rupture occurrence for study population was 3.09 ± 2.43 days.
- 22 (64.7%) patients in FWR subgroup and 19(39.6%) patients in VSR subgroup had rupture (</=2) days after STEMI symptom onset.
- In the present study hypertension 53(64.6%) was the most common co-morbidity among patients of cardiac rupture followed by diabetes 36(43.9%). Majority of patients 80(97.6%) didn't had any prior history of coronary artery disease.
- In the present study, majority 59(72%) of the patients of cardiac rupture were those patients who didn't received any form of reperfusion therapy. 18 (22%) patients developed cardiac rupture after thrombolysis, while 5 (6.1%) patients developed cardiac rupture after PCI, out of which 4 patients received primary PCI.
- In the present study, most common infarction site was anterior wall (41.5%) followed by inferior wall (23.2%). 29 (35.4%) patients had multiple wall (>2 walls) infarction. However, among patients of FWR inferior wall infarction was more common than anterior wall infarction (32.4% vs 26.5%).
- Among patients of VSR apical septal rupture was the most common location seen in 33(68.7%) patients followed by basal septum

rupture seen in 13(27.1%) patients, 2 (4.2%) patients had mid septum rupture.

- Among patients of FWR, acute rupture was seen in 13(38.2%) patients, while 21(61.8%) patients developed subacute free wall rupture.
- In the present study, 19(39.5%) patients with VSR and 11(32.3%) patients with FWR underwent coronary angiography. Overall coronary angiography was performed in only 30(36.6%) patients. 14 (46.7%) patients had SVD, 9 (30%) patients had DVD, 7 (23.3%) patients had TVD.
- In the present study LAD was the most common culprit vessel seen in 18(60%) patients followed by RCA AND LCX in 6(20%) patients each. On subgroup analysis LAD was most common culprit vessel in both subgroups.
- In the present study we found that patients of cardiac rupture have lower blood pressures, the mean Systolic and Diastolic BP were 100.17 ± 29.014 & 62.90 ± 12.455 (mmHg). Patients with cardiac rupture had elevated total leucocyte count (TLC) mean value was 15.34 ± 5.273(10^3/ul), higher creatinine mean creatinine was 1.51 ± 0.949 (mg/dl), elevated cardiac biomarkers mean troponin- T was 3.02 ± 2.131(ng/ml) & elevated natriuretic peptide levels mean BNP was 986.21 ± 896.311(pg/ml) & lower LVEF mean ejection fraction was 33.32 ± 5.204% respectively. On subgroups analysis, we found that patients of VSR had lower mean systolic and diastolic blood pressures & higher mean serum creatinine & BNP levels than FWR patients.
- Cardiac tamponade was seen in 34(100%) patients of FWR. Pericardiocentesis was performed in 12(35.3%) patients of FWR.
- In the present study 46(56.1%) patients were given mechanical circulatory support in the form of IABP (83.3% in VSR vs 17.6% in FWR) subgroup.
- Majority 59 (72%) patients were managed conservatively (62.5% in VSR vs 85.2% in FWR) subgroup. Only 23(28%) patients were

taken for surgical repair (37.5% in VSR vs 14.7% in FWR) subgroup. 5 (6.1%) patients underwent concomitant CABG (8.3% in VSR vs 2.9% in FWR) subgroup along with surgical repair.

- The mean time of surgical repair among patients of VSR from day of admission was 6.77 vs 1.6 days in FWR patients.
- In present study we found in-hospital mortality in VSR patients was 66.7% whereas in-hospital mortality in FWR patients was 67.6%. The overall in- hospital mortality in patients of cardiac rupture was 67.1%.
- In present study we found that conservatively managed patients have higher in-hospital mortality, 79.3% in FWR & 96.6% in VSR subgroup.
- In present study we found that surgery is the cornerstone of treatment in patients of cardiac rupture. In-hospital mortality in surgically managed patients was 0.0% in FWR & 16.6% in VSR subgroup.
- In this study, we found that acute FWR rupture was associated with a higher in-hospital mortality rate compared with subacute. (100% vs 47.7%).
- In the present study out of 21 patients of subacute FWR, 16 (76.2%) patients were conservatively managed. Out of 16 patients managed conservatively 6(37.5%) survived. Hence, we found that a small subset of patients of subacute FWR can be managed conservatively, in whom a small tear might be temporarily sealed by a clot or fibrinous pericardial adhesions that may be compatible with life for several hours or even longer.

- On univariate analysis factors associated with in-hospital survival in patients of cardiac rupture were:
  - Use of IABP (p value =0.032)
  - Surgical repair (p value =0.001)

- On univariate analysis factors associated with in-hospital mortality in patients of cardiac rupture were:

- Age > 70 yrs (p value =0.004))
- Early rupture (</=2days) (p value =0.004).
- Killip class IV on admission (p value =0.035).
- Elevated TLC (10^3/ul) (p value=0.015)
- Higher troponin-T (ng/ml) (p value =0.001)
- Conservative management (p value =0.001)

- On multiple linear regression analysis, we did not find any factor to be significantly associated with in-hospital mortality or survival. This might be due to small sample size.

CONCLUSION

Out of 82 patients of cardiac rupture, 48 had ventricular septum rupture while 34 had free wall rupture. Patients with cardiac rupture are more likely to be elderly with age > 65yrs, hypertensive, have no prior history of coronary artery disease, first MI, usually anterior wall MI, have single vessel disease, have lower rate of primary percutaneous coronary intervention (PPCI), and have higher in-hospital mortality. Compared with survivors, non survivors are older >70yrs, have early occurrence of cardiac rupture(</=2days), are in killip class IV on admission, have elevated white blood cell count & higher troponin-T, and lower rate of surgical repair. Role of IABP in survival among patients of cardiac rupture remains debatable. Surgical repair remains the most valuable factor associated with survival in patients of cardiac rupture.

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KEY TO MASTER CHART

Clinical Presentation at the time of hospitalization

1 - Angina

2 - Cardiogenic Shock

3 - Cardiac Arrest

Reperfusion Therapy

1 - None

2 - Thrombolysis

3 - PCI

Myocardial Infarction Site

1 - Anterior

2 - Inferior

3 - Multiple (> 2 walls)

Type of cardiac rupture

1 - Acute LV Free wall Rupture

2 - Sub-Acute Free wall Rupture

3 - Ventricular Septal Rupture (Apical Septum)

4 - Ventricular Septal Rupture (Mid Septum)

5 - Ventricular Septal Rupture (Basal Septum)

Coronary Angiography

1 - SVD (Single Vessel Disease)

2 - DVD (Double Vessel Disease)

3 - TVD (Triple Vessel Disease)

Culprit Vessel

1 - LAD (Left Anterior Descending Artery)

2 - RCA (Right Coronary Artery)

3 - LCX (Left Circumflex Artery)

SR. NO.	Name	Age (years)	Sex	MRD. No	Past Medical history												Time from symptom onset to hospitalisatiion (Hours)	Clinical Presentation at the time of hospitalization	SBP (mmHg)	DBP (mmHg)	Pulse (bpm)	Killip Class	Timing of Cardiac Rupture Diagnosis (Days)	Reperfusion Therapy	Baseline investigations						
					Diabetes mellitus	Hypertension	Hypercholestrolemia	Smoking	CAD	PCI	CABG	Stroke /TIA	PAD	CKD	COPD										Hemoglobin (gm/dL)	TLC (10^3/ul)	Platelets (10^3/ul)	Creatinine (mg/dL)	Troponin – T (ng/mL)	BNP (pg/mL)	PCT (ng/mL)
1	JS	57	Male	1131751	Yes	Yes	Yes	No	No	No	No	No	No	No	No	5	1	130	80	102	2	4	2	16.7	22.4	203	1.4	1.89	199	0.05
2	NL	62	Male	1228772	No	Yes	No	No	No	No	No	No	No	No	No	36	1	100	60	100	3	2	2	12.8	17.6	237	1.2	7.81	200	0.05
3	RK	65	Male	1207482	Yes	Yes	No	Yes	No	No	No	No	Yes	No	No	2	2	80	60	158	4	1	1	11.2	23	454	1.6	2.69	702	8.01
4	VK	52	Male	430691	No	Yes	No	No	No	No	No	No	No	Yes	No	48	2	80	60	112	4	3	2	13.2	21	249	7.31	3.45	1800	0.5
5	PK	71	Female	892075	No	Yes	No	No	No	No	No	No	No	Yes	No	3	2	Not Recordable	Not Recordable	122	4	1	1	9.1	16.8	291	4.9	3.65	1510	0.05
6	AS	51	Male	315287	No	Yes	No	Yes	No	No	No	No	No	No	No	10	2	90	60	124	4	1	2	15.6	18.5	261	1	1.24	1010	7.8
7	GS	50	Male	987707	No	No	No	No	No	No	No	No	No	No	No	96	2	80	60	138	4	5	1	13	19.4	322	1	1.34	1832	0.05
8	KK	55	Female	327245	Yes	Yes	No	No	No	No	No	No	No	No	No	28	2	Not Recordable	Not Recordable	110	4	2	1	9.4	16.4	248	1.2	0.34	1234	1.62
9	LB	65	Male	328520	No	Yes	No	Yes	No	No	No	No	No	No	Yes	48	2	80	50	84	4	3	1	10.4	9.3	608	0.7	0.43	1189	0.08
10	JS	55	Male	321766	Yes	Yes	No	No	No	No	No	No	No	No	No	1	2	Not Recordable	Not Recordable	102	4	1	1	12.5	10.9	239	1	2.14	103	0.05
11	TS	83	Male	374546	No	No	Yes	Yes	No	No	No	No	No	No	No	96	2	80	60	102	4	5	1	12.4	15.9	192	1.5	1.89	1456	0.05
12	VP	65	Male	454535	Yes	No	No	No	No	No	No	No	No	No	No	16	2	90	60	122	4	1	1	12.4	7.5	194	1.9	1.02	1412	0.05
13	KK	66	Female	1196470	No	Yes	No	No	No	No	No	No	No	No	No	26	2	90	60	88	4	2	1	9.1	18.7	142	1.8	2.1	1640	0.3
14	PDS	67	Male	495973	No	No	No	No	No	No	No	No	No	No	No	4	2	Not Recordable	Not Recordable	116	4	1	1	13.1	11.6	210	1.59	2.62	200	0.05
15	HK	60	Female	356941	No	Yes	No	No	No	No	No	No	No	No	No	240	1	110	70	84	3	11	1	8.5	16	271	2	1.36	1700	2.23
16	KK	61	Female	379344	Yes	Yes	Yes	No	No	No	No	Yes	No	No	No	28	2	90	60	104	4	2	2	9.2	21.7	427	1.37	0.34	1234	0.05
17	MK	52	Female	630277	Yes	Yes	No	No	No	No	No	No	No	No	No	72	2	90	60	86	4	4	2	13.2	11.1	305	1.06	1.13	1283	0.08
18	DK	61	Female	957295	Yes	Yes	No	No	No	No	No	No	No	No	No	18	2	Not Recordable	Not Recordable	94	4	1	1	9.4	21.6	140	1.9	1.67	1468	0.05
19	PO	59	Female	806506	Yes	No	No	No	No	No	No	No	No	Yes	No	28	2	80	60	128	4	2	1	12.7	23.6	162	2	2.42	1378	0.05
20	SH	74	Female	737302	Yes	No	Yes	No	No	No	No	No	No	No	No	48	1	100	60	110	3	3	1	11.2	23.3	356	1.3	2.3	3980	0.9
21	RS	69	Male	815327	No	No	No	No	Yes	No	Yes	No	Yes	No	No	24	2	Not Recordable	Not Recordable	58	4	2	1	10.1	15.7	140	2.7	3.1	2200	0.05
22	SK	58	Female	933469	Yes	Yes	Yes	No	No	No	No	No	No	No	No	48	2	80	60	143	4	3	1	12.6	16.5	273	1.1	2.65	400	0.05
23	DS	65	Male	879394	No	No	Yes	Yes	No	No	No	No	No	No	No	48	2	90	60	104	4	3	2	13.6	9.8	301	1	3.47	835	0.05
24	KS	75	Male	885861	No	No	Yes	Yes	No	No	No	No	No	No	No	72	1	110	60	110	3	4	2	14	14.8	408	1.2	0.97	1290	0.6
25	GS	70	Male	730754	No	Yes	No	No	No	No	No	No	No	Yes	No	36	2	90	70	114	4	2	2	14.6	23.6	214	3.3	1.79	1467	1.76
26	RS	66	Male	1042382	Yes	No	No	Yes	No	No	No	No	No	No	No	72	1	100	70	98	3	4	1	14.3	11.2	120	1.2	0.78	1232	0.75
27	BS	64	Male	909638	No	No	No	No	Yes	Yes	No	No	No	No	No	86	2	80	56	120	4	4	1	13.2	12.8	252	1.1	0.34	880	0.05
28	SR	54	Female	957722	Yes	Yes	Yes	No	No	No	No	No	No	Yes	No	76	2	80	60	78	4	4	1	10.2	8	240	1.4	0.96	649	0.05
29	HR	62	Male	1429838	No	Yes	Yes	Yes	No	No	No	No	No	No	No	8	2	80	60	114	4	1	1	13.2	13.1	272	1.1	2.6	130	1.8
30	RK	66	Male	975898	Yes	No	No	Yes	No	No	No	No	No	No	No	72	2	80	60	84	4	4	1	12.3	16	130	1.4	1.76	240	0.05
31	UG	59	Female	1432590	No	No	Yes	No	No	No	No	No	No	No	No	24	1	130	90	74	1	5	3	14.6	7.6	259	0.6	0.38	113	0.05
32	KK	72	Female	1196576	Yes	No	Yes	No	No	No	No	No	No	Yes	No	8	2	80	60	68	4	1	2	11.8	18.6	237	1.9	6.13	311	0.05
33	RK	80	Female	1121392	Yes	Yes	Yes	No	No	No	No	Yes	No	No	No	6	1	124	88	56	1	2	2	13	18.3	467	1.1	0.61	200	0.05
34	RK	65	Male	1227357	Yes	No	No	Yes	No	No	No	No	No	No	No	26	2	70	60	112	4	2	1	13.7	16.3	140	2.94	4.84	1719	0.05
35	JS	61	Male	1206675	No	Yes	No	No	No	No	No	No	No	No	No	10	2	90	50	92	4	1	2	14.2	16.8	279	1.2	3.12	612	0.05
36	US	82	Male	1209119	No	No	No	No	No	No	No	No	No	No	No	28	2	80	60	50	4	2	1	11.2	16.7	215	1.04	6.23	220	0.05
37	SD	70	Female	1220835	Yes	No	No	No	No	No	No	No	No	No	No	80	2	88	50	96	4	4	1	11.1	10.9	276	1.3	2.1	894	0.05
38	KA	62	Male	1223914	No	Yes	No	No	No	No	No	No	No	No	No	96	1	170	80	98	1	5	2	10.9	7.7	174	1	1.05	289	0.07
39	MD	69	Female	1164706	No	Yes	No	No	No	No	No	No	No	No	No	48	2	80	60	120	4	3	1	12.2	13	276	1.5	4.5	2060	0.05
40	SL	62	Female	1228573	Yes	Yes	No	No	No	No	No	No	No	No	No	46	2	80	50	116	4	3	1	12.6	18.5	185	2.4	2.54	2200	0.05
41	HK	60	Female	1120066	Yes	No	No	No	No	No	No	No	No	No	No	96	1	102	68	124	2	5	1	12.4	13.5	265	1.4	1.53	2280	0.05
42	MS	75	Male	1241087	Yes	Yes	No	No	No	No	No	No	No	No	No	6	1	150	100	102	1	1	2	17.9	11.4	204	1	6.2	69	0.05
43	RAJU	46	Male	1258289	No	No	No	Yes	No	No	No	No	No	No	No	26	2	60	40	120	4	2	1	16	23	259	0.9	5.54	400	0.05
44	PL	58	Male	1272992	No	Yes	No	No	No	No	No	No	No	No	No	8	1	100	70	96	2	3	3	14	11.6	99	0.9	1.28	649	0.05

SR. NO.	Cholesterol (mg/dL)	Myocardial Infarction Site	Ejection Fraction (%)	Type of cardiac rupture	Pericardial Tamponade	Coronary Angiography	Culprit Vessel	Medication on Admission									Treatment							Outcome
																		IABP	PCI	Concomitant CABG		Sugical repair		
								Aspirin	P2Y12 Inhibitor	Beta - Blocker	ACE- Inhibitor	LMWH	Diuretics	Statins	Amiodarone	Ionotropes	Pericardiocentesis	(Yes/No)	(Yes/No)	(Yes/No)	Day	(Yes/No)	Day	
1	276	1	28	2	Yes	1	1	Yes	Yes	No	No	Yes	Yes	Yes	No	No	No	No	No	No	-	No	-	Discharge
2	188	1	32	2	Yes	-	-	Yes	Yes	No	No	Yes	No	Yes	No	Yes	No	No	No	No	-	No	-	Death
3	163	2	25	5	No	-	-	Yes	No	No	No	Yes	No	Yes	No	Yes	No	Yes	No	No	-	No	-	Death
4	149	1	30	3	No	-	-	Yes	No	No	No	Yes	Yes	Yes	No	Yes	No	Yes	No	No	-	No	-	Death
5	91	2	35	5	No	-	-	Yes	Yes	No	No	Yes	Yes	Yes	Yes	Yes	No	Yes	No	No	-	No	-	Death
6	155	3	32	3	No	-	-	Yes	No	No	No	Yes	Yes	Yes	No	Yes	No	Yes	No	No	-	No	-	Death
7	181	3	36	5	No	-	-	Yes	Yes	No	No	Yes	Yes	Yes	No	Yes	No	Yes	No	No	-	No	-	Death
8	132	2	28	5	No	1	2	Yes	Yes	No	No	Yes	No	Yes	No	Yes	No	Yes	No	No	-	Yes	12	Discharge
9	176	3	32	3	No	1	1	Yes	No	No	No	Yes	Yes	Yes	Yes	Yes	No	Yes	No	No	-	Yes	5	Discharge
10	161	2	30	2	Yes	2	3	No	No	No	No	No	No	No	No	Yes	No	Yes	No	No	-	Yes	1	Discharge
11	146	1	32	3	No	1	1	Yes	No	No	No	Yes	Yes	Yes	No	Yes	No	Yes	No	No	-	Yes	7	Discharge
12	162	1	36	3	No	-	-	Yes	No	No	No	Yes	Yes	Yes	Yes	Yes	No	Yes	No	No	-	No	-	Death
13	188	1	28	3	No	-	-	Yes	No	No	No	Yes	No	Yes	No	Yes	No	Yes	No	No	-	No	-	Death
14	182	3	36	2	Yes	3	1	No	No	No	No	No	No	Yes	No	Yes	No	Yes	No	Yes	5	Yes	1	Discharge
15	105	1	30	3	No	2	1	Yes	No	No	No	Yes	No	Yes	No	Yes	No	Yes	No	No	-	Yes	8	Discharge
16	224	3	35	3	No	2	1	Yes	Yes	No	No	Yes	No	Yes	No	Yes	No	Yes	No	No	-	Yes	7	Discharge
17	170	1	34	3	No	1	1	Yes	No	No	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes	10	Yes	10	Discharge
18	174	1	28	4	No	-	-	Yes	No	No	No	No	No	Yes	No	Yes	No	No	No	No	-	No	-	Death
19	88	2	38	5	No	-	-	Yes	No	No	No	Yes	No	Yes	No	Yes	No	Yes	No	No	-	No	-	Death
20	191	1	35	3	No	-	-	Yes	No	No	No	Yes	Yes	No	No	Yes	No	No	No	No	-	No	-	Death
21	124	1	32	3	No	-	-	Yes	Yes	No	No	Yes	Yes	Yes	No	Yes	No	No	No	No	-	No	-	Death
22	242	3	36	2	Yes	-	-	No	No	No	No	No	No	No	No	Yes	No	Yes	No	No	-	Yes	1	Discharge
23	197	1	38	3	No	2	1	Yes	No	No	No	Yes	No	Yes	No	Yes	No	Yes	No	No	-	Yes	9	Discharge
24	316	3	32	3	No	2	1	Yes	No	No	No	Yes	Yes	Yes	No	Yes	No	Yes	No	Yes	6	Yes	6	Death
25	130	3	45	5	No	3	2	Yes	No	No	No	Yes	No	Yes	No	Yes	No	Yes	No	Yes	7	Yes	7	Death
26	137	1	28	3	No	1	1	Yes	No	No	No	Yes	Yes	Yes	No	Yes	No	Yes	No	No	-	Yes	6	Discharge
27	145	3	34	3	No	-	-	Yes	No	No	No	Yes	No	Yes	No	Yes	No	Yes	No	No	-	Yes	4	Discharge
28	198	1	42	3	No	-	-	Yes	No	No	No	Yes	Yes	Yes	Yes	Yes	No	Yes	No	No	-	No	-	Death
29	168	2	32	2	Yes	2	3	No	No	No	No	No	No	Yes	No	Yes	Yes	Yes	No	No	-	No	-	Discharge
30	93	2	36	2	Yes	1	3	No	No	No	No	No	No	Yes	No	Yes	No	Yes	No	No	-	Yes	1	Discharge
31	243	3	48	1	Yes	1	1	Yes	Yes	No	No	Yes	No	Yes	No	No	Yes	No	No	No	-	No	-	Death
32	199	2	32	2	Yes	-	-	Yes	Yes	No	No	Yes	No	Yes	No	Yes	No	No	No	No	-	No	-	Death
33	227	1	38	2	Yes	-	-	Yes	Yes	No	No	Yes	No	Yes	No	No	No	No	No	No	-	No	-	Death
34	212	3	36	5	No	-	-	Yes	Yes	No	No	Yes	No	Yes	No	Yes	No	No	No	No	-	No	-	Death
35	168	3	34	5	No	-	-	Yes	No	No	No	Yes	No	Yes	No	Yes	No	No	No	No	-	No	-	Death
36	154	2	32	1	Yes	-	-	Yes	Yes	No	No	Yes	No	Yes	No	Yes	No	No	No	No	-	No	-	Death
37	164	1	40	3	No	1	1	Yes	Yes	No	No	Yes	No	Yes	No	Yes	No	Yes	No	No	-	Yes	6	Discharge
38	104	3	28	2	Yes	3	1	Yes	Yes	No	Yes	No	No	Yes	No	No	Yes	No	No	No	-	No	-	Discharge
39	146	1	33	3	No	-	-	Yes	Yes	No	No	Yes	Yes	Yes	No	Yes	No	No	No	No	-	No	-	Death
40	118	3	36	5	No	-	-	Yes	Yes	No	No	Yes	Yes	Yes	No	Yes	No	Yes	No	No	-	No	-	Death
41	193	3	34	4	No	2	1	Yes	No	No	No	Yes	No	Yes	No	Yes	No	Yes	No	No	-	Yes	4	Discharge
42	166	3	36	1	Yes	-	-	Yes	No	No	No	Yes	No	Yes	No	No	No	No	No	No	-	No	-	Death
43	196	2	26	2	Yes	-	-	No	No	No	No	No	No	Yes	No	Yes	No	No	No	No	-	No	-	Death
44	139	3	34	3	No	1	3	Yes	Yes	No	No	Yes	No	Yes	No	Yes	No	No	No	No	-	No	-	Discharge

SR. NO.	Name	Age (years)	Sex	MRD. No	Past Medical history											Time from symptom onset to hospitalisatiion (Hours)	Clinical Presentation at the time of hospitalization	SBP (mmHg)	DBP (mmHg)	Pulse (bpm)	Killip Class	Timing of Cardiac Rupture Diagnosis (Days)	Reperfusion Therapy	Baseline investigations						
					Diabetes mellitus	Hypertension	Hypercholestrolemia	Smoking	CAD	PCI	CABG	Stroke /TIA	PAD	CKD	COPD									Hemoglobin (gm/dL)	TLC (10^3/ul)	Platelets (10^3/ul)	Creatinine (mg/dL)	Troponin – T (ng/mL)	BNP (pg/mL)	PCT (ng/mL)
45	VAS	54	Male	126016	No	No	No	No	No	No	No	No	No	No	No	48	2	60	40	142	4	3	1	14.4	20	220	1.7	4.54	237	0.05
46	KB	68	Female	1253050	No	Yes	No	No	No	No	No	No	No	No	No	72	1	100	60	112	3	4	1	10.5	18.3	315	1.1	1.48	1120	0.33
47	BV	56	Male	1337929	Yes	Yes	No	No	No	No	No	No	No	No	No	8	1	170	80	96	1	2	2	13.8	9.6	207	1	0.586	12	0.22
48	AS	90	Male	578099	No	Yes	No	No	No	No	No	No	No	No	Yes	14	1	100	60	110	2	1	1	12.8	6.5	252	1.3	5.28	1640	1.02
49	JS	53	Male	1255777	Yes	No	No	No	No	No	No	No	No	No	No	168	1	130	80	88	2	14	1	12.3	8.6	124	1.2	2.28	581	0.73
50	SK	65	Female	1304290	Yes	No	No	No	No	No	No	No	No	Yes	No	240	1	100	60	89	3	12	1	10.6	12.9	110	3.9	3.68	5000	0.96
51	SS	70	Male	1292807	No	No	No	No	No	No	No	No	No	No	No	48	1	100	60	98	3	4	1	9.7	6.2	204	1	1.71	1130	0.05
52	CL	75	Male	1290906	Yes	Yes	No	No	No	No	No	No	No	No	No	28	2	80	60	96	4	2	1	13.3	11.4	280	1.7	7.37	2450	0.05
53	GS	77	Male	1287374	No	Yes	No	No	No	No	No	No	No	No	No	3	1	140	80	84	1	1	2	12.3	14.8	210	1.1	5.48	200	0.05
54	JR	75	Female	1297396	No	Yes	No	No	No	No	No	No	No	No	No	4	1	130	70	98	1	1	2	9.9	11.6	272	0.9	5.78	98	0.05
55	HK	80	Female	129927	No	Yes	No	No	No	No	No	No	No	Yes	No	4	1	90	69	84	4	1	3	9.5	11.7	178	1.8	4.48	680	0.05
56	WC	71	Male	1331202	Yes	Yes	No	No	No	No	No	No	No	No	No	96	2	80	50	110	4	5	1	12.9	18.4	181	1.8	5.76	1860	0.05
57	AK	68	Female	1352405	Yes	Yes	No	No	No	No	No	No	No	No	No	92	2	70	50	110	4	5	1	11.8	22.3	210	1.8	4.56	1478	0.05
58	JK	63	Female	1333671	No	Yes	No	No	No	No	No	No	No	No	No	52	1	180	60	122	1	3	1	8.4	12.5	394	0.6	1.53	204	0.05
59	SK	71	Male	253223	Yes	Yes	No	No	No	No	No	No	No	No	No	48	2	90	60	79	4	3	1	13.1	11.4	188	1.7	3.64	660	0.54
60	SK	69	Female	1398354	No	Yes	No	No	No	No	No	No	No	No	No	48	1	100	60	110	3	3	1	11	14.4	243	1	4.98	1260	0.05
61	AK	75	Female	1406710	No	Yes	No	No	No	No	No	No	No	No	No	4	2	90	60	116	4	1	1	13.5	19.1	122	1.1	1.46	128	0.05
62	NKS	68	Female	1404584	No	Yes	No	No	No	No	No	No	No	No	Yes	14	2	60	40	64	4	1	1	12.1	9.6	307	1.73	0.473	175	0.05
63	GK	61	Female	1391327	No	Yes	No	No	No	No	No	No	No	No	No	52	2	90	60	109	4	3	1	12	23.3	312	1.5	2.13	2200	0.5
64	TMS	80	Male	140382	No	Yes	No	No	No	No	No	No	No	No	No	72	1	150	70	110	1	5	1	14.5	11.3	146	1	3.27	148	0.05
65	KS	62	Male	1289762	Yes	No	No	No	No	No	No	No	No	No	No	18	1	100	60	42	3	5	1	13.1	22.1	168	2.1	8.23	1650	2.96
66	AK	68	Female	1352405	Yes	Yes	Yes	No	No	No	No	No	No	No	No	96	2	60	40	110	4	5	1	11.8	22.8	210	1.8	4.13	1822	1.93
67	KL	72	Female	1366582	Yes	Yes	No	No	No	No	No	No	No	No	Yes	3	3	Not Recordable	Not Recordable	136	4	1	1	12.6	11.4	210	1.2	7.68	200	0.05
68	SK	60	Male	1353722	Yes	Yes	No	No	No	No	No	No	No	No	No	46	3	Not Recordable	Not Recordable	148	4	2	1	15.5	12.9	161	1.9	4.65	1760	0.05
69	MK	75	Female	1381389	No	Yes	Yes	No	No	No	No	No	No	No	No	3	1	70	50	94	4	1	1	11	14.4	244	1.4	1.84	200	0.05
70	SS	85	Male	1405422	No	Yes	No	No	No	No	No	No	No	No	No	3	1	160	80	87	1	1	1	12.2	12.9	244	1.1	4.56	101	0.05
71	UK	80	Female	1212371	No	No	No	No	No	No	No	No	No	No	No	4	1	130	70	86	1	2	3	10.9	11.5	317	0.7	0.268	143	0.05
72	RG	61	Male	858661	No	No	Yes	Yes	No	No	No	No	No	No	No	24	1	140	80	82	1	4	3	15.9	14.4	166	0.89	1.21	79	0.05
73	AS	65	Male	1370191	Yes	No	Yes	No	No	No	No	No	No	No	No	72	2	82	42	122	4	4	1	12.9	16.5	336	0.9	2.79	790	0.05
74	PH	42	Male	1377640	No	Yes	Yes	Yes	No	No	No	No	No	No	Yes	8	1	180	90	82	1	4	2	14.8	16.9	153	0.6	0.128	64	0.05
75	SD	57	Female	1350823	Yes	Yes	No	No	No	No	No	No	No	No	No	72	1	110	60	110	3	5	1	9.5	10.2	219	0.9	2.89	1583	0.05
76	DK	63	Male	663225	No	Yes	No	Yes	No	No	No	No	No	No	No	22	1	100	58	84	3	2	1	10	9.5	216	1	2.94	811	0.74
77	JSR	62	Male	1346919	No	No	No	No	No	No	No	No	No	No	No	168	1	122	88	112	2	10	1	12.9	9.1	410	0.9	0.614	194	0.07
78	SK	60	Female	987006	Yes	No	No	No	No	No	No	No	No	Yes	No	28	2	Not Recordable	Not Recordable	84	4	2	1	11.9	35.9	178	2.1	6.78	1840	16.85
79	AK	73	Female	1246808	No	Yes	No	No	No	No	No	No	No	No	No	24	2	84	54	112	4	2	1	10.8	19.6	200	1.5	3.87	760	0.05
80	SR	73	Female	863557	Yes	Yes	No	No	No	No	No	No	No	No	No	18	2	Not Recordable	Not Recordable	112	4	2	1	12	20.8	166	1.5	3.99	416	4.24
81	KJ	78	Female	1433797	No	Yes	Yes	No	No	No	No	No	No	No	No	8	3	Not Recordable	Not Recordable	98	4	1	1	11.5	14.5	210	1.2	6.58	242	0.05
82	LR	60	Male	1280818	No	Yes	Yes	Yes	No	No	No	No	No	No	Yes	1	3	Not Recordable	Not Recordable	68	4	1	1	14.6	16.8	310	1	7.65	84	0.05

SR. NO.	Cholesterol (mg/dL)	Myocardial Infarction Site	Ejection Fraction (%)	Type of cardiac rupture	Pericardial Tamponade	Coronary Angiography	Culprit Vessel	Medication on Admission									Treatment							Outcome
																	Pericardiocentesis	IABP	PCI	Concomitant CABG		Sugical repair		
								Aspirin	P2Y12 Inhibitor	Beta - Blocker	ACE- Inhibitor	LMWH	Diuretics	Statins	Amiodarone	Ionotropes		(Yes/No)	(Yes/No)	(Yes/No)	Day	(Yes/No)	Day	
45	103	2	35	2	Yes	-	-	Yes	No	No	No	No	No	No	No	Yes	Yes	No	No	No	-	No	-	Discharge
46	159	2	34	5	No	2	2	Yes	No	No	No	Yes	No	Yes	No	Yes	No	Yes	No	No	-	Yes	5	Discharge
47	140	2	42	5	No	3	2	Yes	Yes	No	No	Yes	No	Yes	No	No	No	Yes	No	No	-	Yes	5	Discharge
48	141	1	38	3	No	1	1	Yes	No	No	No	Yes	Yes	Yes	No	Yes	No	Yes	No	No	-	No	-	Death
49	111	2	32	2	Yes	-	-	Yes	Yes	No	No	Yes	No	Yes	No	No	Yes	No	No	No	-	No	-	Discharge
50	95	1	36	3	No	1	1	Yes	Yes	No	No	Yes	Yes	Yes	No	Yes	No	Yes	No	No	-	Yes	9	Discharge
51	95	2	36	5	No	3	2	Yes	Yes	No	No	Yes	Yes	Yes	No	No	No	Yes	No	Yes	6	Yes	6	Discharge
52	123	2	34	5	No	-	-	Yes	Yes	No	No	Yes	No	Yes	No	Yes	No	Yes	No	No	-	No	-	Death
53	126	1	42	1	Yes	-	-	Yes	Yes	No	No	Yes	No	Yes	No	No	No	No	No	No	-	No	-	Death
54	153	3	34	1	Yes	-	-	Yes	No	No	No	Yes	No	Yes	No	No	No	No	No	No	-	No	-	Death
55	166	2	32	1	Yes	2	2	Yes	Yes	No	No	Yes	No	Yes	No	Yes	Yes	No	No	No	-	No	-	Death
56	192	3	28	3	No	-	-	Yes	Yes	No	No	Yes	No	Yes	No	Yes	No	Yes	No	No	-	No	-	Death
57	153	1	32	3	No	-	-	Yes	Yes	No	No	Yes	No	Yes	No	Yes	No	Yes	No	No	-	No	-	Death
58	169	3	36	2	Yes	-	-	Yes	No	Yes	Yes	Yes	No	Yes	No	No	No	Yes	No	No	-	No	-	Death
59	158	3	24	3	No	1	3	Yes	No	No	No	Yes	No	Yes	No	Yes	No	Yes	No	No	-	Yes	6	Death
60	115	1	26	3	No	-	-	Yes	Yes	No	No	Yes	No	Yes	No	Yes	No	Yes	No	No	-	No	-	Death
61	128	3	36	2	Yes	-	-	No	No	No	No	No	Yes	Yes	No	Yes	Yes	No	No	No	-	No	-	Death
62	147	2	42	2	Yes	-	-	Yes	Yes	No	No	Yes	No	Yes	No	Yes	No	No	No	No	-	No	-	Death
63	184	1	24	3	No	-	-	Yes	Yes	No	No	Yes	Yes	Yes	Yes	Yes	No	Yes	No	No	-	No	-	Death
64	146	2	34	1	Yes	-	-	Yes	No	Yes	Yes	Yes	No	Yes	No	No	No	No	No	No	-	No	-	Death
65	167	1	22	3	No	-	-	Yes	Yes	No	No	Yes	Yes	Yes	No	Yes	No	Yes	No	No	-	No	-	Death
66	153	1	24	3	No	-	-	Yes	Yes	No	No	No	No	No	No	Yes	No	Yes	No	No	-	No	-	Death
67	212	3	24	2	Yes	-	-	No	No	No	No	No	No	No	No	Yes	Yes	No	No	No	-	No	-	Death
68	187	1	28	3	No	-	-	Yes	Yes	No	No	Yes	No	Yes	Yes	Yes	No	Yes	No	No	-	No	-	Death
69	111	1	36	2	Yes	-	-	No	No	No	No	No	No	Yes	No	Yes	No	No	No	No	-	No	-	Death
70	119	3	42	1	Yes	-	-	Yes	Yes	Yes	Yes	Yes	No	Yes	No	No	No	No	No	No	-	No	-	Death
71	156	1	24	1	Yes	1	1	Yes	Yes	No	No	Yes	No	Yes	No	No	Yes	No	No	No	-	No	-	Death
72	269	1	42	1	Yes	3	1	Yes	No	Yes	No	Yes	No	Yes	No	No	Yes	No	No	No	-	No	-	Death
73	194	1	32	3	No	-	-	Yes	Yes	No	No	Yes	No	Yes	No	Yes	No	Yes	No	No	-	No	-	Death
74	182	3	36	2	Yes	-	-	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Yes	No	Yes	No	No	No	-	No	-	Discharge
75	136	1	25	3	No	-	-	Yes	Yes	No	No	Yes	Yes	Yes	No	No	No	Yes	No	No	-	No	-	Death
76	97	3	34	2	Yes	-	-	Yes	Yes	No	No	Yes	Yes	Yes	No	Yes	No	No	No	No	-	No	-	Death
77	124	3	38	2	Yes	3	3	Yes	Yes	No	No	Yes	No	Yes	No	No	No	No	No	No	-	Yes	4	Discharge
78	173	1	32	3	No	-	-	Yes	Yes	No	No	Yes	No	Yes	No	Yes	No	Yes	No	No	-	No	-	Death
79	148	3	32	3	No	-	-	Yes	Yes	No	No	Yes	No	Yes	No	Yes	No	No	No	No	-	No	-	Death
80	133	3	32	1	Yes	-	-	Yes	Yes	No	No	Yes	Yes	Yes	No	Yes	Yes	No	No	No	-	No	-	Death
81	186	1	32	1	Yes	-	-	No	No	No	No	No	No	No	No	Yes	No	No	No	No	-	No	-	Death
82	192	1	38	1	Yes	-	-	No	No	No	No	No	No	No	No	Yes	No	No	No	No	-	No	-	Death

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The Principal,
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Subject: **Approval of Plan of Thesis.**

This is to inform you that the Faculty of Medical Sciences in its meeting held on 29.10.2021 has approved the plan of thesis of **Amit Kumar Gupta**, a student of your college for **DM Cardiology** along with the names of Supervisor/Co-Supervisor as under:-

Topic Plan of Thesis
Clinical presentation, management strategies, and in-hospital outcome in cardiac rupture.

Supervisor
Dr. Shibba Takkar Chhabra
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Co-Supervisor

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The student has to submit the four copies of thesis (along with PDF format) , to the Controller of Examinations, BFUHS, Faridkot, at least six months before the commencement of theory examination, if he/she is otherwise eligible to take the examination. He /She must directed to attach a photocopy of this letter with the forwarding letter while submitting thesis to the University along with the letter of approval of University for change of supervisor/Co-Supervisor, change of topic,(if any).

Dr. S P Singh MS(Oph)
Controller of Examinations

Note :

153. *If any discrepancy found regarding topic of thesis or Supervisor/Co-Supervisor, inform undersigned within one month after issue of this letter.*
154. *The student may submit the thesis as per the schedule already conveyed i.e for May/June examination 30th Nov of the preceding the year of examination and for Nov/Dec examinations 31st May of the year of examination.*

CLINICAL PRESENTATION, MANAGEMENT STRATEGIES, AND IN-HOSPITAL OUTCOME IN CARDIAC RUPTURE

THESIS PROPOSAL

FOR APPROVAL OF SUBJECT OF THESIS TO BE SUBMITTED

IN PARTIAL FULFILMENT OF THE REQUIREMENTS

FOR THE DEGREE OF

DOCTORATE OF MEDICINE

IN

CARDIOLOGY

OF THE

BABA FARID UNIVERSITY OF HEALTH SCIENCES
FARIDKOT

JULY, 2021

DR AMIT KUMAR GUPTA

DEPARTMENT OF CARDIOLOGY
DAYANAND MEDICAL COLLEGE & HOSPITAL
LUDHIANA

ABSTRACT OF PLAN OF THESIS

Title	CLINICAL PRESENTATION, MANAGEMENT STRATEGIES, AND IN-HOSPITAL OUTCOME IN CARDIAC RUPTURE
For the degree of	D.M Cardiology
Name of the candidate	Dr Amit Kumar Gupta
Supervisor	Dr Shibba Takkar Chhabra, Professor, Department of Cardiology, DMCH, Ludhiana
Co-supervisor	Dr Naved Aslam , Professor, Department of Cardiology, DMCH, Ludhiana Dr. Gurbhej Singh, Assistant professor, Department of Cardiology, DMCH, Ludhiana
Institution	Dayanand Medical College & Hospital, Ludhiana
University	Baba Farid University of Health Sciences, Faridkot

Introduction: Following cardiogenic shock and fatal ventricular arrhythmias, cardiac rupture is ranked third as the leading cause of all infarct-related deaths. The advent of primary percutaneous interventions compared to the pre-thrombolytic or the thrombolytic era, has considerably reduced the rates of cardiac rupture. However, the mortality continues to remain high.

Aim of Study: 1. To study the clinical presentation in patients of cardiac rupture.

2. To evaluate the management strategies and in-hospital outcome in patients of cardiac rupture.

Materials & methods: The study will be conducted in department of cardiology at Dayanand Medical College and Hospital, Ludhiana (DMC&H).

Study Design:

It will be a observational study done in retrospective and prospective manner to study the clinical presentation and evaluate the management strategies and in-hospital outcome in patients of cardiac rupture.

Clinical Significance: Cardiac rupture due to variable clinical presentation and high mortality (75%-90%) remains a substantial diagnostic and therapeutic challenge for clinicians. The diagnosis of cardiac rupture can be made accurately using bedside echocardiography, as long as clinicians are aware of the risk factors, clinical signs and other subtle manifestations. Moreover, the development of surgical repair has improved the prognosis of patients with cardiac rupture. So, timely diagnosis and emergent interventions can save patient's lives.

Keywords: LVFWR, AMI, STEMI, PCI.

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CERTIFICATE OF FACILITIES AVAILABLE

This is to certify that the facilities for work on the subject of thesis titled **"CLINICAL PRESENTATION, MANAGEMENT STRATEGIES, AND IN-HOSPITAL OUTCOME IN CARDIAC RUPTURE"** exist in the Department of Cardiology, DMC&H, and Ludhiana and will be provided to the candidate. We will see that the data being included in the thesis is genuine and is collected by the candidate himself under our supervision and guidance.

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6 BRIEF RESUME OF THE INTENDED WORK

6.1 NEED FOR THE STUDY

Cardiac rupture is a rare but fatal complication of AMI. It accounts for approximately 20% of STEMI patient in-hospital deaths.[1] The three types of cardiac rupture are ventricular septal rupture, free-wall rupture, and papillary muscle rupture.

During the era of thrombolytic therapy, or the no reperfusion era, the incidence of cardiac rupture after STEMI was 6%.[2] In recent years, the incidence of cardiac rupture after AMI has decreased to less than 2% with the widespread use of PPCI.[3]

The mortality rate for free-wall rupture is 75% to 90%, and the mortality rate for ventricular septal rupture is 40% to 75%.[4]

Risk factors for cardiac rupture include advanced age, female gender, absence of prior angina, history of hypertension, no history of myocardial infarction, anterior infarct, absence of PPCI, single-vessel disease without collateral circulation, anemia, and elevated heart rate.[3,4,5]

The clinical presentation varies from a catastrophic blowout type characterized by cardiogenic shock and eventually cardiac arrest, to the oozing type with haemodynamic instability and pericardial effusion.[6]

Several studies have tried to describe the warning signs and symptoms of fatal cardiac rupture.[7,8,9-11] Prodromal manifestations include persistent chest pain, intractable vomiting, restlessness, also electrocardiogram (ECG) signs as persistent S-T segment elevation, and positive T wave deflection that persists for 72 hours after the onset of chest pain.[7,9] Other classic signs of cardiac tamponade, including pulsus paradoxus and diastolic pressure equalization, may not exist in most of the cases.[10]

Echocardiography is the most critical tool for a timely diagnosis with a sensitivity and specificity of more than 90%. The median time of death following LVFWR is 8 hours.[12]

Prompt diagnosis and management can lead to successful treatment for cardiac rupture. Conservative strategies have been described,[13,14]

but the poor results of this treatment make surgical intervention generally mandatory. Despite different surgical approaches having been proposed, in-hospital mortality continues to be high[15,16] and the most appropriate surgical management remains still controversial, particularly in terms of rupture recurrence or other type of post-operative complications.

Overall, cardiac rupture due to the variable clinical presentation and associated high mortality remains a substantial diagnostic and therapeutic challenge for clinicians.

The diagnosis of cardiac rupture can be made accurately using bedside echocardiography, as long as clinicians are aware of the risk factors, clinical signs and other subtle manifestations, such as ECG changes and the development of surgical repair has improved the prognosis of AMI patients with cardiac rupture.

So, Timely diagnosis is key to successful emergent interventions that save patients' lives.

Thus, this study aimed to evaluate the clinical presentation, management strategies, and in-hospital outcomes in patients of cardiac rupture with AMI .

6.2 REVIEW OF LITERATURE

History: The first free-wall rupture of the heart after AMI was described by William Harvey in 1647.[17] In 1769, Morgagni collected and reported 11 cases of myocardial rupture discovered postmortem.[18] Ironically, Morgagni himself later died of ventricular rupture. However, it was not until 1925, in the report of Krumbhaar and Crowell, that the relationship of LVFWR to myocardial infarction was first pointed out.[19]

Hatcher and associates, from Emory University, in 1970 described the first successful operation for free-wall rupture of the right ventricle.[20] Two years later, Fitzgibbon et al. reported the successful repair of LVFWR associated with ischemic heart disease.[21]

Pathophysiology of cardiac rupture:

It involves thinning of the myocardial wall with the intensity of necrosis occurring at the distal end of the vessel (watershed area) where there is often poor collateral flow. The shearing effect of myocardial contraction against a stiffened necrotic area causes a rupture.

Clinically cardiac ruptures can be divided into:

Acute rupture which results in death within a few minutes due to massive haemorrhage into the pericardial cavity; Subacute rupture which is characterized by a smaller tear which may be temporarily sealed by a clot or fibrinous pericardial adhesions which may be compatible with life for several hours or even longer; Chronic rupture with false aneurysm formation which occurs when the leakage of blood is slow and when surrounding pressure on the epicardium temporarily controls the hemorrhage. Adhesions form between the epicardium and pericardium which reinforce and contain the rupture.

Ruptures have also been classified as early when it develops within the first 48 hours or late rupture when it develops beyond the second day.

Factors facilitating free wall rupture include delayed hospital admission >12–24 hours, persistent systemic hypertension, unusual in hospital physical effort (agitation, repetitive vomiting, coughing), extension of myocardial infarction, expansion of myocardial infarction.[22]

Free wall rupture may occur within 24 hours or between 5–7 days but rupture has been reported to occur as late as one month or even beyond.[23] Previous literature studies report the commonest site of rupture to be the anterior wall. Recent series have observed greater occurrence of lateral and posterior wall ruptures.[24]

Figueras J et al[2] in 2008 investigated the incidence of cardiac rupture in ST-elevation myocardial infarction patients and its mortality rate over 30-year period. Of a total of 6678 consecutive patients, 425 experienced a free wall rupture (280 with cardiac tamponade: 227 with electromechanical dissociation and 53 with hypotension) or a septal rupture (145). After the exclusion of referrals from other centers (n=44), the incidence of definite cardiac rupture (septal rupture, anatomic evidence of free wall rupture, or electromechanical dissociation) declined progressively in parallel with a progressive use of reperfusion. In addition, among patients with cardiac rupture, there was a progressive fall in the rate of death despite a trend toward increasing age in conjunction with better control of systolic blood pressure at 24 hours, an increased use of reperfusion therapy, beta-blockers, angiotensin-converting enzyme inhibitors, and aspirin and a lower use of heparin.

Global Registry of Acute Coronary Events (GRACE) [25] Investigators in 2010 studied factors related to heart rupture in acute coronary syndromes in 60198 patients and reported that, 273 had Heart Rupture (free wall rupture in 118 and ventricular septal rupture in 155). Incidence was 0.9% for ST-segment elevation myocardial infarction (STEMI), 0.17% for non-STEMI, and 0.25% for unstable angina. Hospital mortality was 58 vs. 4.5% in patients without heart rupture. The incidence was lower in STEMI patients with primary percutaneous coronary intervention (PCI) than in those without, but primary PCI was not independently related to heart rupture. Independent variables associated with heart rupture included: ST-segment elevation (STE)/left bundle branch block; ST-segment deviation; female sex; previous stroke; positive initial cardiac biomarkers; older age; higher heart rate; systolic blood pressure/30

mmHg decrease. Conversely, previous MI and the use of low-molecular-weight heparin and beta-blockers during first 24 h were identified as protective factors for heart rupture.

Honda S et al [3] in 2014 evaluated the trends in the Clinical and Pathological Characteristics of cardiac rupture in Patients With Acute Myocardial Infarction over 35 years. Of a total of 5699 patients, 144 were diagnosed with cardiac rupture and 45 survived. Over the years, the incidence of cardiac rupture decreased in association with the widespread adoption of reperfusion therapy. The mortality rate of cardiac rupture decreased in association with an increase in the rate of emergent surgery. In multivariable analysis, first myocardial infarction, anterior infarct, female sex, hypertension, and age >70 years were significant risk factors for cardiac rupture, whereas impact of hypertension on cardiac rupture was weaker from 2001 to 2011. Primary percutaneous coronary intervention (PPCI) was a significant protective factor against cardiac rupture. In 64 autopsy cases with cardiac rupture, myocardial hemorrhage occurred more frequently in those who underwent PPCI or fibrinolysis than those who did not receive reperfusion therapy.

Nasir et al [13] in 2014 reviewed 120 papers and concluded that surgery is superior to conservative management for patients presenting with free wall rupture. However, Che J et al[26] in 2016 stated that conservative management can be successfully carried out in elderly patients and patients at a high surgical risk, who have small infarction with slow bleeding and absence of other mechanical complications.

Leitman M [27] et al in 2016 evaluated 2,380 patients admitted to Assaf Harofeh Medical Center with the diagnosis of acute myocardial infarction between January 2008 and February 2015 and reported that 12 (0.5%) developed myocardial rupture. The mean age was 78 years, and there were 7 males and 5 females. Ten patients already had pericardial effusion on admission. Seven patients underwent coronary angiography, whilst primary percutaneous intervention was performed in 4 patients. Six patients entered the operating room and all survived the procedure. All patients who were treated conservatively died due to

rupture. Factors related to the treatment strategy were advanced age (≥ 90 years) and cognitive impairment. The risk of myocardial rupture may be diminished by primary coronary intervention during myocardial infarction, but mortality remains high. An early, comprehensive echocardiographic examination and rapid surgery may contribute to improved survival.

Formica F et al [15] in 2018 conducted a retrospective study of 35 patients who underwent surgery for LVFWR between January 2000 and December 2016. The mean age of patients was 68.3 years. The in-hospital survival was 65.7%, and 13% of survived patients presented with cardiac arrest. On univariable analysis: pre-existing hypertension, need for inotropes, and cardiac arrest at presentation, cardiopulmonary resuscitation, preoperative extracorporeal membrane oxygenation, technique of LVFWR repair operation on extracorporeal membrane oxygenation and postoperative extracorporeal membrane oxygenation were associated with in-hospital mortality . On multivariable analysis, cardiac arrest at presentation was an independent predictor of in-hospital mortality. The overall mean postoperative follow-up was 8.3 years. Overall survival rates at 5 and 10 years were 53.2 % and 49.1%, respectively. Among the survivors, only 6 (26.1%) patients died during follow-up with a 5-year and 10-year overall survival rate of 80.9 % and 74.7%, respectively.

Varghese S et al [28] in 2019 retrospectively studied 5143 patients with AMI at the Central Hospital, Bad Berka. Out of these, seven patients with LVFWR were identified with an overall incidence of 0.14%. Clinically, LVFWR patients presented late to admission since symptom onset (24 h), were more likely in cardiogenic shock (28.6%) and were usually accompanied by emergency physicians (71.4%). Higher troponin T (8.6), higher CRP (50) as well as a lower hematocrit-values (0.33) were observed. All LVFWR patients were operated (100%). The patients had lower rates of beta-blocker treatment (57.1%). The 30-day mortality was significantly higher (42.9%).

Hao Z et al [1] in 2020 retrospectively analysed cardiac rupture in 4190 ST-elevation myocardial infarction patients from 2004 to 2015 at

Renji Hospital, School of Medicine, Shanghai Jiaotong University and reported that out of 4190 patients, 75 (1.8%) patients had cardiac rupture, 33 had ventricular septum rupture while 42 had left ventricle free wall rupture (LVFWR). Patients with cardiac rupture were more likely to be female, with more advanced age, lower rate of primary percutaneous coronary intervention (PPCI), and higher in-hospital mortality. Compared with survivors, the death cases were older, had a higher white blood cell count, higher rate of delayed admission (>12 h from symptom onset to door), earlier occurrence of cardiac rupture, higher percentage of LVFW rupture and lower rate of surgical repair. Logistic regression analysis showed that surgical repair served as the most valuable factor affecting survival. Moreover, elevated white blood cell count and advanced age might be related to an increased in-hospital death due to cardiac rupture.

6.3 AIMS AND OBJECTIVES OF THE STUDY

- To study the clinical presentation in patients of cardiac rupture.
- To evaluate the management strategies and in-hospital outcome in patients of cardiac rupture.

7.0 MATERIALS AND METHODS

The study will be conducted in department of cardiology at Dayanand Medical College and Hospital, Ludhiana (DMC&H).

Study Design:

It will be a observational study done in retrospective and prospective manner to study the clinical presentation and evaluate the management strategies and in-hospital outcome in patients of cardiac rupture.

7.1 SOURCE OF DATA

For retrospective analysis, data of all consecutive patients with cardiac rupture admitted in DMC&H during last 10 years will be taken from institutional data base.

For prospective analysis, informed consent will be taken from patient or patient's attendant presenting to emergency department of DMC&H with cardiac rupture from 1st August 2021 to 31 December 2022. Patients will be assessed on basis of history, physical examination, investigation and treatment received.

7.2 METHOD OF COLLECTION OF DATA

Inclusion Criterion:

- All patients of acute of myocardial infarction presenting to DMC&H with cardiac rupture on admission or diagnosed to have cardiac rupture during hospital stay.

Exclusion Criterion:

- Lack of informed consent.
- Patients presenting with cardiac rupture from causes other than myocardial infarction.
- Patients presenting with papillary muscle rupture.

The diagnosis of STEMI will be based on typical clinical symptoms, electrocardiographic (ECG) findings of ST elevation .1 mV in more than two limb leads or .0.2 mV in two or more contiguous precordial leads, as well as documented cardiac enzyme (troponin I or T) elevation

according to the definition of myocardial infarction from the Fourth Universal Definition of Myocardial Infarction.

Cardiac rupture will be diagnosed on 2D-Echocardiographic / cardiac CT or MRI findings.

All patients who fulfills the criteria will be evaluated and the data will be collected in case record form.

OUTCOME: The primary outcome will be in-hospital mortality. Secondary outcome will be factors associated with in-hospital mortality in patients of cardiac rupture.

STATISTICAL ANALYSIS: Data will be described in terms of range; mean ± standard deviation (± SD), frequencies (number of cases) and relative frequencies (percentages) as appropriate. To determine whether the data were normally distributed, a Kolmogorov-Smirnov test was used. Comparison of quantitative variables between the study groups will be done using Student t-test and Mann Whitney U test for independent samples for parametric and non-parametric data respectively. For comparing categorical data, Chi square ($\chi 2$) test will be performed and exact test was used when the expected frequency is less than 5. A probability value (p value) less than 0.05 will be considered statistically significant. All statistical calculations will be done using (Statistical Package for the Social Science) SPSS 21version (SPSS Inc., Chicago, IL, USA) statistical program for Microsoft Windows.

7.3 DOES THE STUDY REQUIRE ANY INVESTIGATIONS OR INTERVENTIONS TO BE CONDUCTED ON PATIENTS OR OTHER HUMANS OR ANIMALS? IF SO, PLEASE DESCRIBE BRIEFLY.

NO

7.4 HAS ETHICAL CLEARENCE BEEN OBTAINED FROM YOUR INSTITUTION IN CASE OF 7.3?

ATTACHED HEREWITH

8.0 LIST OF REFERENCES

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PROFORMA

Sr. No

Name: **Age / Sex:** **MRD. No.**

D.O.A:

Address:

PHYSICAL EXAMINATION:

Height(cm): **Weight(kg):** **B.P (mm hg):** **Pulse(per/min) :**

RR(per/min): **BMI:**

JVP: **Pallor:** **Icterus:** **Cyanosis :**

Clubbing : **Edema:** **LAP:**

CARDIOVASCULAR EXAMINATION

	Positive findings	
Inspection		
Palpation		
Auscultation	Aortic area	Pulmonary area
	Mitral area	Tricuspid area

Associated risk factors	**Present**	**Absent**
Diabetes mellitus		
Hypertension		
Obesity		
Smoking		
Other Co-morbidities		

Signature of Candidate **Signature of Supervisor**

Previous History of	Present	Absent
IHD (LMCA/ SVD/ DVD/TVD)		
Thrombolysis		
PTCA		
CABG		
Other cardiac disease		

Previous medication history		
Antiplatelets		
Beta blockers		
Ace inhibitors / ARBs		
Aldosterone antagonists		
Diuretics		
Statins		
Other medications		

Time from symptoms onset to hospitalization (Hours/ Days):

Direct Admission / referred:

Clinical Presentation at the time of hospitalization: Angina / Cardiogenic Shock / Cardiogenic Arrest / Others

Timing of cardiac rupture	Early (<48 hours)	Late (After 48 hours)
Post acute myocardial		

Signature of Candidate **Signature of Supervisor**

infarction		
Post thrombolysis		
Post PTCA(Stent thrombosis)		
Post CABG		

Investigations:

Baseline investigations	**At time of admission**	**At time of diagnosis of rupture**	**At time of discharge**
Hemoglobin			
TLC			
Platelets			
Creatinine			
Troponin –I / T			
CRP			
BNP			
PCT			
Total Cholesterol			

ECG(myocardial infarction site) : Inferior/ anterior / Lateral / Posterior /multiple wall infarction

(two or more)

2D ECHOCARDIOGRAPHY/: Cardiac rupture: (Free wall / ventricular septal)

Type of cardiac rupture (Acute/ subacute/ chronic)

(Blow out / oozing)

Signature of Candidate **Signature of Supervisor**

Location of rupture:

CLOT: (Present/ Absent)

Pericardial effusion: (Size/ Location/ Loculated or Diffuse)

Cardiac Tamponade -Diastolic collapse of RA/RV (Present/ Absent)

IVC (mm) -Size (Dilated/ collapse)

Ejection fraction(%)

Other Findings **:**

Cardiac CT/MRI (if available):

Coronary angiography (Culprit vessel): LMCA LAD LCx RCA

Management: Conservative management: Ionotropes

IABP

Pericardiocentesis

ECMO

Surgical management: Patch Repair + Glue

Direct Suture

± (PTCA/ CABG)

± Additional Surgical Procedure

Signature of Candidate **Signature of Supervisor**

Outcome: Death

Survive

Medical follow up (if available):

Signature of Candidate **Signature of Supervisor**

INFORMED PATIENT CONSENT

Date: _____

I……..D/o, W/o, S/o……. ……am willing to participate in the study **"CLINICAL PRESENTATION, MANAGEMENT STRATEGIES, AND IN-HOSPITAL OUTCOME IN CARDIAC RUPTURE"** The procedure with its complications has been explained to me in local vernacular language. I have fully understood that no special tests and extra expenditure in any form, merely for the purpose of thesis is being carried out. I am free to opt out the study any time without assigning any reason.

……. ……

Signature of the Witness Signature of the patient

Name:

Signature of Candidate **Signature of Supervisor**

मरीज अनुमति फार्म

तिथि _____

मैं _____ पत्नी/पुत्र/पुत्री _____

निवासी _____

इस शोधकार्य "**CLINICAL PRESENTATION, MANAGEMENT STRATEGIES, AND IN-HOSPITAL OUTCOME IN CARDIAC RUPTURE"** में भाग लेने के लिए सहमत हूँ। मैं इस शोधकार्य में भाग लेने के लिए अपनी स्वेच्छा से तैयार हूँ। मैं पुष्टि करता/करती हूँ की यह स्वीकृति देने के लिए मुझे कोई आर्थिक प्रलोभन नहीं दिया गया और न ही दबाव डाला गया है। मुझे इस शोधकार्य का उद्देश्य स्पष्ट कर दिया गया है और मुझे यह विश्वास है की यह विज्ञान और मानवता के लाभ के लिए है। मुझे इस शोधकार्य में होने वाले जोखिम के बारे में समझा दिया गया है और मेरी समझ में यह जोखिम बहुत मामुली है।

मैं इस शोधकार्य में भाग लेने के सम्बन्ध में किसी भी समय अपनी अस्वीकृति व्यक्त कर सकता/सकती हूँ और मेरे अस्वीकृति व्यक्त करने का मेरे इलाज पर कोई प्रभाव नहीं पड़ेगा।

_____ _____

गवाह के हस्ताक्षर रोगी के हस्ताक्षर/अंगूठे का निशान

नाम: नाम:

_____

अन्वेषक के हस्ताक्षर

_____

विद्यार्थी के हस्ताक्षर

Signature of Candidate **Signature of Supervisor**

ਮਰੀਜ਼ ਰਜ਼ਾਮੰਦੀ ਫਾਰਮ

ਮਿਤੀ_____

ਮੈ_____ਪਤਨੀ/ਪੁੱਤਰ/ਪੁੱਤਰੀ_____

ਵਾਸੀ _____

“CLINICAL PRESENTATION, MANAGEMENT STRATEGIES, AND IN-HOSPITAL OUTCOME IN CARDIAC RUPTURE” ਤੇ ਚਲ ਰਹੀ ਖੋਜ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਰਜ਼ਾਮੰਦ ਹਾਂ। ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਲਈ ਮੈ ਆਪਣੀ ਮਰਜ਼ੀ ਨਾਲ ਤਿਆਰ ਹਾਂ। ਮੈ ਇਹ ਦਸਣਾ ਚਾਹੁੰਦਾ ਹਾਂ/ ਚਾਹੁੰਦੀ ਹਾਂ ਕੇ ਇਹ ਰਜ਼ਾਮੰਦੀ ਦੇਣ ਵਾਸਤੇ ਮੈਨੂ ਕਿਸੇ ਵੀ ਤਰਾਂ ਦਾ ਲੋਭ-ਲਾਲਚ ਨਹੀ ਦਿੱਤਾ ਗਿਆ ਅਤੇ ਨਾ ਹੀ ਕੋਈ ਦਬਾ ਪਾਇਆ ਗਿਆ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਦਾ ਮਨੋਰਥ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਭਰੋਸਾ ਦਵਾਇਆ ਗਿਆ ਹੈ ਕੇ ਇਹ ਖੋਜ ਵਿਗਿਆਨ ਅਤੇ ਮਾਨਵਤਾ ਦੀ ਭਲਾਈ ਲਈ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਵਿਚ ਹੋਣ ਵਾਲੇ ਖਤਰੇ ਬਾਰੇ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ ਅਤੇ ਮੈਨੂ ਇਹ ਯਕੀਨ ਹੈ ਕੇ ਇਹ ਖਤਰਾ ਬਹੁਤ ਮਾਮੂਲੀ ਹੈ। ਮੈਨੂ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਦਾ ਢੰਗ – ਤਰੀਕਾ ਸਮਝਾ ਦਿੱਤਾ ਗਿਆ ਹੈ।

ਮੈ ਇਸ ਖੋਜ ਕੰਮ ਵਿਚ ਹਿੱਸਾ ਲੈਣ ਬਾਰੇ ਆਪਣੀ ਰਜ਼ਾਮੰਦੀ ਕਿਸੇ ਵੀ ਸਮੇ ਵਾਪਸ ਲੈ ਸਕਦਾ ਹਾਂ/ ਸਕਦੀ ਹਾਂ ਅਤੇ ਮੇਰੀ ਰਜ਼ਾਮੰਦੀ ਵਾਪਸ ਲੈਣ ਦਾ ਮੇਰੇ ਇਲਾਜ ਤੇ ਕੋਈ ਅਸਰ ਨਹੀ ਹੋਵੇਗਾ।

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ਗਵਾਹ ਦੇ ਹਸਤਾਖਰ ਮਰੀਜ਼ ਦੇ ਹਸਤਾਖਰ

ਮਿਤੀ_____ ਮਿਤੀ_____

ਪਤਾ _____ ਪਤਾ _____

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ਨਿਰੀਖਕ ਦੇ ਹਸਤਾਖਰ

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ਸਿਖਿਆਰਥੀ ਦੇ ਹਸਤਾਖਰ

Signature of Candidate **Signature of Supervisor**